DOES NOT CIRCULATE

Journal of

Forensic Medicine

UNIVERSITY OF MICHIGAN
MAR 16 1955
MEDICAL LIBRARY

Tardieu Spots in Asphyxia—EDITORIAL 1

Carbon Monoxide Poisoning—KEITH SIMPSON 5

How Trustworthy is the Witness?—HENRY A. DAVIDSON 14

Cross-Examination: Its Use and Abuse—WARREN P. HILL 21

Subpleural, Subpericardial and Subendocardial Haemorrhages—I. GORDON, R. A. MANSFIELD, J. E. DUNCAN TAYLOR, K. A. B. KOHLER, I. PRINSLOO, I. ROBERTSON AND L. G. VON THOMANN 31

Foetal Sciatic Notch: Sex Differences—BARBARA J. BOUCHER 51

Manganese in Human Tissues. I: Its Determination—P. R. v. d. R. COPEMAN 55

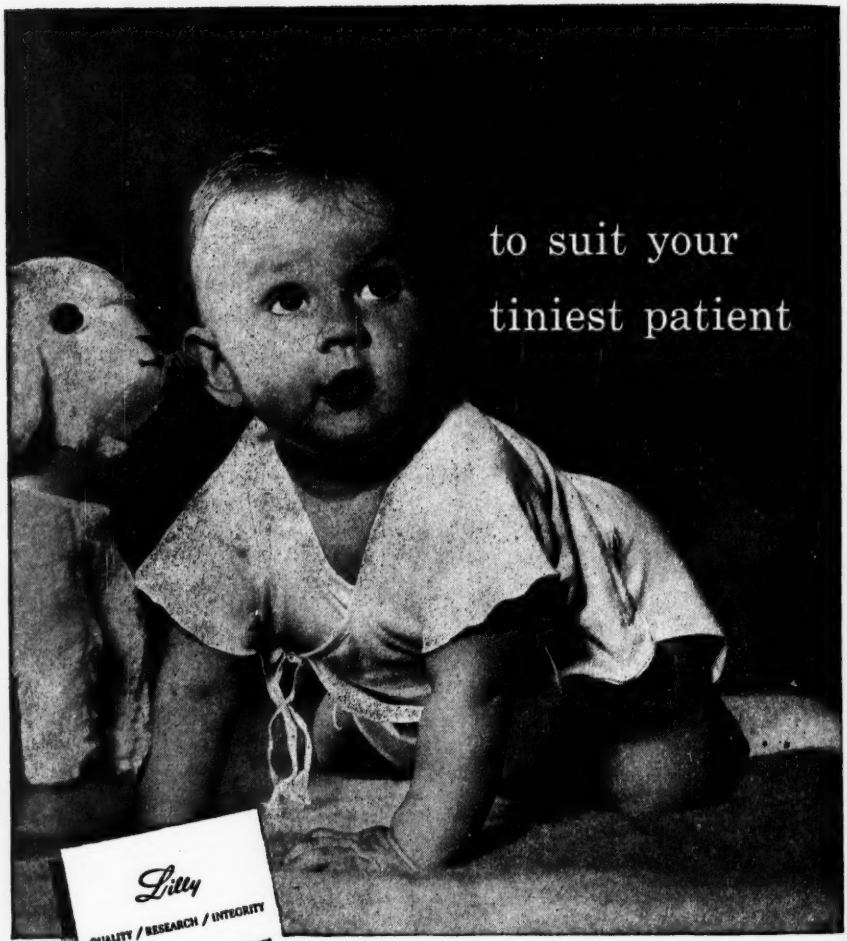
Reviews of Books 58

Correspondence: Breath Tests for Alcohol—R. N. HARGER 62

VOL. 2: NO. 1: JANUARY-MARCH 1955: PRICE 12/6: ANNUAL SUBSCRIPTION 42/-

Published Quarterly by Juta & Co. Ltd.,

P.O. Box 30, Cape Town : P.O. Box 1010, Johannesburg



ILOTYCIN DROPS

(ERYTHROMYCIN, LILLY) ETHYL CARBONATE

with an unexcelled antibiotic spectrum

another reason to consider ILOTYCIN FIRST

- Appeals to the physician for its effectiveness and safety
- Appeals to the nurse and mother for its convenience in administration
- Appeals to baby for its taste-tested flavor

ELI LILLY INTERNATIONAL CORPORATION, INDIANAPOLIS 6, INDIANA, U.S.A.

Sole Distributors: Ethical Products (Pty.) Ltd., the Ethical Division of Johnson & Johnson (Pty.) Ltd., P.O. Box 727, East London.

Journal of Forensic Medicine

P.O. Box 1010 · Johannesburg · South Africa

Sponsored by the South African Medico-Legal Society

Editor: H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.Af.

Tardieu Spots in Asphyxia

In the nineteenth century Tardieu described minute haemorrhages due to rupture of small blood vessels on the surfaces of the lungs, the heart and other parts of the body. He regarded them as characteristic of death from suffocation. They have since eponymously come to be known as Tardieu spots and have long been regarded as diagnostic of all forms of asphyxiation due to mechanical obstruction of the airway.

Tardieu's claims were sufficiently novel to provoke opposition at the time in Germany; and the proceedings in one of the most notable trials of the century (that of the body-snatchers Burke and Hare) are of great interest in connexion with his views on asphyxia.

Dr. Robert Christison, then Professor of Medical Jurisprudence in Edinburgh University, did the autopsy on one of Burke's victims alleged to have been murdered (by suffocation) to provide anatomical material for Dr. Knox. Professor Christison did not observe, or at all events describe, the small haemorrhages regarded as so characteristic of asphyxia, especially in the internal organs. His evidence [1] is characterized by a reluctance to appear dogmatic. Indeed, he stated: 'I never would give my opinion ultimately and decisively upon a case of this kind, without inquiring into the collateral circumstances I have mentioned' (p. 195).

Nevertheless, over the years the asphyxial significance of Tardieu's small haemorrhages came to be accepted by successive generations of medico-legal examiners of dead bodies. This is reflected in contemporary writings. Taylor,[2] e.g. states that their presence 'suggests some form of asphyxial

1. Roughead, W. Ed. by (1948): *Trials of Burke and Hare (Notable British Trials Series)*, 3rd ed., p. 195. London: W. Hodge and Company, Limited.
2. Smith, S. Ed. by (1948): *Taylor's Principles and Practice of Medical Jurisprudence*, 10th ed., 1, 524. London: J. and A. Churchill, Ltd.

death'. Smith and Fiddes[3] regard them as almost constant in 'young children dying from asphyxia . . . and even when there is no air in the lungs (they) are evidence of attempted respiration'. Keith Simpson[4] apparently considers they provide some of the necessary evidence of the lethal consequences which follow the asphyxiating obstruction.

Most standard textbooks imply that an asphyxial mode of death can be inferred confidently from the post-mortem evidences observed in the internal organs alone. Some, indeed, profess to make a pathological distinction between asphyxia, coma and syncope and have even described a 'mixed' form of death, viz. comato-asphyxia.[5] The error inherent in this approach is partly due to a confusion between clinical states, observed in the living, and their counterpart in the dead, where different criteria apply. This problem has been reviewed in an Editorial in an earlier issue of this *Journal*.[6]

Gordon[7] has already drawn attention to the fact that the so-called asphyxial changes seen in the internal organs occur in many deaths (including those due to natural causes), thus confirming the experience of other careful observers. The signs in the internal organs are often dominated by Tardieu spots and Gordon has grouped them together as the basic or general signs found in deaths from almost any cause. They cannot, therefore, be invested with the diagnostic significance hitherto attributed to them. This has led to emphasis on the medico-legal importance of the *local* (as opposed to the *general*) pathological signs seen in those dead from asphyxia.

In many asphyxial deaths there is actual and good evidence of the way in which the obstruction of the airway was produced. In the overlaying of infants (the category originally studied by Tardieu) there may be flattening of the tissues about the nose and the mouth. In throttling, finger nail abrasions on the neck, with additional injury to the skin and the deeper structures, will give good evidence of the site of application of the force.

In strangling there may be the evidence of the constricting ligature and its mark. In drowning, the presence of the fluid medium in the air passages and the stomach assumes significance. These are all evidences in a localized part of the body indicative of the way in which the obstructive, asphyxiating process operated. They must be distinguished clearly from the non-specific general signs which may be present in varying degree. Indeed,

3. Smith, S. and Fiddes, F. S. (1949): *Forensic Medicine*, 9th ed., p. 251. London: J. and A. Churchill, Ltd.
4. Simpson, Keith (1954): In *The British Encyclopaedia of Medical Practice: Medical Progress Volume*, p. 109. London: Butterworth Medical Publications.
5. Glaister, J. (1950): *Medical Jurisprudence and Toxicology*, 9th ed., p. 145. Edinburgh: E. and S. Livingstone, Ltd.
6. Editorial (1953): *Is Asphyxia a Pathological Entity Recognizable Post Mortem?* J. Forensic Med., 1, 65.
7. Gordon, I. (1944): *A Classification of Deaths of Medico-Legal Importance*, Brit. Med. J., 2, 337.

it is primarily from these local signs, together with the history of the case, that a reasonably certain inference can be drawn about the mode of death, provided the autopsy discloses no evidence of disease or other injury incompatible with life.

Keith Simpson [4] has objected that the local signs, e.g. in strangling, afford evidence only of the application of some form of constriction. They are not evidence of the lethal effects which follow the constriction. He regards the Tardieu spots, *inter alia,* as such evidence. Yet haemorrhages of this type can occur clinically in non-fatal conditions, e.g. whooping cough, in the 'asphyxial' distribution, viz. the eyes, the face and the neck.

This view also overlooks the fact that the medico-legal pathologist is not always able to state categorically the cause of death, which may have to be a reasonable inference from all the relevant facts (as Professor Christison appreciated with such wisdom in 1828). Cases of asphyxia, when they have to be diagnosed from the findings in the dead body alone, may often place the pathologist in a difficult position, which he should never regard as embarrassing merely because the case for the prosecution is not thereby strengthened. His concern is always only with the warranted inferences that scientifically acceptable data permit to be drawn. Whether these are helpful to the prosecution should be a matter of complete indifference to him.

In any event, the value of this search for the lethal evidences of asphyxia is questioned severely by the publication (elsewhere in this issue) of the most recent contribution from Gordon and his co-workers. This carefully controlled quantitative study establishes that so-called Tardieu spots can occur for the first time after death. They are true minute haemorrhages which arise spontaneously *post mortem* on the very surfaces inspected for the alleged diagnostic evidences of asphyxia. This clearly indicates that they are entirely unconnected with an asphyxial mode of production in the living; and that they cannot have necessary significance as a lethal sign in the dead, thus destroying what force there may have been in the suggestion that Tardieu's haemorrhagic spots are evidence of a fatal asphyxia.

Moreover, in some instances, what looked like true haemorrhages to the naked eye have been seen to disappear. Microscopic examination confirmed that these were not haemorrhages at all, but merely dilatations of minute blood vessels, simulating Tardieu spots.

It therefore becomes essential, in any case of forensic importance where the pathologist still attaches weight to these Tardieu spots, to obtain microscopic verification that they are actually haemorrhages; but even this is a labour lost, since the researches of Gordon and his co-workers indicate that there is no means of telling whether the haemorrhages occurred shortly before death, agonally during the process of dying or after death. Nor is the number, size or colour of the spots of any assistance. Even their distribution may not be of much help as they can arise for the first time in non-

dependent areas such as the front of the heart, when the body is lying on its back.

This raises the importance of the position of the body after death in connexion with the significance of Tardieu spots in other parts, e.g. the skin of the face and neck. Strassmann[8] has frequently seen small haemorrhages (of the type claimed as characteristic of asphyxia) on the back of the body in areas of post-mortem lividity, to which the blood has gravitated (p. 132). He states that these haemorrhages can likewise occur in the face without preceding violence, if the victim's head was prone after death (p. 276).

Gonzales *et al.*[9] also describe such haemorrhages occurring after death in dependent areas of lividity and actually call them Tardieu spots, thereby clearly recognizing that they do not necessarily have ante-mortem significance.

The many reservations in the past about the diagnostic significance of Tardieu spots have thus been more than justified. This is not to say they do not occur before death, whether in asphyxia or other states. Indeed, if they are confined to a region above a ligature round the neck, and if the possible effects of posture can be excluded, they may well be regarded as having the significance of a local sign, albeit somewhat superfluous in the presence of the ligature. But the force of the earlier criticism, that too much importance has been attached to signs in the internal organs, is much enhanced by the important observations of Gordon and his co-workers. In addition to their non-specificity, the spots have now been found to be without necessary validity as an ante-mortem event. This reinforces the importance of the local evidences in the study of asphyxial modes of death and places the haemorrhagic spots in the internal organs in their proper, insignificant perspective.

There seems much merit in Christison's nineteenth century caution which insisted on adequate inquiry into all the collateral circumstances in these cases.

8. Strassmann, G. (1954): In *Legal Medicine*, ed. by R. B. H. Gradwohl, pp. 132, 276. St. Louis: C. V. Mosby Co.
9. Gonzales, T. A., Vance, M., Helpern, M. and Umberger, C. J. (1954): *Legal Medicine, Pathology and Toxicology*, 2nd ed., p. 52. New York: Appleton-Century-Crofts, Inc.

Carbon Monoxide Poisoning

Medico-Legal Problems

Keith Simpson, M.D. (Path.) Lond.*

With the Technical Assistance of

R. Furbank

Department of Forensic Medicine, Guy's Hospital, London

In a recent review of the common domestic dangers of CO poisoning in the Metropolis of London (Simpson[5]) attention was drawn to the ease with which the condition might be overlooked altogether—in spite of the exercise of reasonable vigilance. In a series of 100 successive cases of *accidental* poisoning by CO from various sources, no less than 46 were unrecognized until autopsy. The toll of life from this cause is appallingly high (400 to 500 deaths per year in England and Wales alone) and it may well be that even this figure under-estimates the frequency. The reasons for believing such a figure to represent less than the actual total of deaths are cogent enough:

(a) *The circumstances of death* by no means always indicate even the possibility, far less the likelihood, of CO exposure. In 10 of the sample 100 cases of accidental exposure reviewed, no gas or other source of poisonous fumes appeared to be available in the premises when, upon autopsy, it became clear that the victims had succumbed to CO. The gas had made its way into premises devoid of a supply or a stove from an unsuspected outside source.

A woman of 73 and her daughter of 37 were found dead in bed on the ground floor of a house 110 feet distant from a fractured gas pipe under the street pavement. A hole had been burned in the 4-inch main by an electric cable fusing. There was no gas supply in the house, nor any other source for CO poisoning: it had not been suspected. Saturations of 60% and 68% HbCO (respectively) were measured.

In another case a 66-year-old night watchman to premises having no supply of gas was found dead, it was presumed of natural causes. Autopsy revealed signs of CO absorption, confirmed by analysis (HbCO 56%) and investigation resulted in the discovery of a fractured 4-inch main 60 feet away on the opposite side of the street.

* University Reader in Forensic Medicine, Guy's Hospital, London.

Intensely cold weather had caused strain on the cast iron pipe—an increasingly common danger in London's network of elderly service mains—and fracture.

(b) *Deficient sense of smell* accounts for many cases being unsuspected when first found. When police and doctors arrive to investigate, the atmosphere has commonly become cleared by ventilation. In 17 of the cases analysed witnesses admitted at inquest to an impaired sense of smell. No doubt, some similar proportion of the dead suffered from the same dangerous defect: it is far more common than is generally supposed.

Dr. Eric Gardner and I went, in 1946, to an address in Watford, Herts, one evening to investigate the mysterious deaths of a child of 3 years and its grandmother of 58, together with the collapse, and vomiting, of the mother aged 27—all found 'unconscious' in a closed kitchen. A P.C. left on guard for 2½ hours was found holding his helmet, mopping a sweating pallid brow, looking very seedy and complaining of the stuffy atmosphere, of feeling giddy and sick. A coke-burning stove which was still alight was found to have 2 sanitary pads blocking the flue plate. No one had smelt fumes.

(c) *Disease, senescence and drunkenness* may all too easily be assumed to have been responsible for death—when, in fact, only contributing to the circumstances of exposure.

A man of 60, under treatment for Hodgkin's disease, was found dead sitting in his chair by a gas fire: it was unlit and, some ventilation being present, no smell of coal-gas was reported. At autopsy the colour of the blood was thought to be suspiciously pink. Analysis showed a saturation of 40% HbCO, and a visit to the chair in which deceased sat made it clear that one only had to stretch one's feet to kick on a tap by the fireside: it was not of the drop-type. The doctor in this case was prepared to certify death as due to Hodgkin's disease, but had not seen him for 'about a month'.

A woman of 77 was admitted to hospital and treated as a case of cerebral haemorrhage in view of her doctor's stating that she had had two previous strokes and was again unconscious. The physical signs were ill defined. The fact that she had been found in bed with her aged husband (whom examination showed to be dead) had been disregarded. Autopsy revealed a blood saturation of 27% HbCO in the old man—who was a chronic bronchitic subject with a cor pulmonale. The woman recovered although no specific treatment for CO poisoning was undertaken: her intellect was somewhat impaired.

(d) *Failure to think of the possibility of CO poisoning* is, of course, as commonly responsible for missed cases of CO poisoning as of any other disease. None of 6 deaths in the bathroom was suspected to be due to fumes, nor 3 coke stove cases where, it might be thought, the possibility presented itself strongly enough.

Defective ventilation, accounting for 17 of the cases analysed, is commonly either under-estimated or overlooked altogether as a source of danger; 66% of English low-income-group bathrooms, most ventilated by single cowled shafts, have a cubic capacity of 350 cubic feet or less, many of less than 250 cubic feet. American apartment rooms in the same social grade often house unventilated refrigerators allowed to 'burn dirty'.[4] Adequate ventilation is essential both to proper combustion and to safety. The same is, of course, true of the small kitchen, the stoke-hole, the garage, the caravan or the ship's galley: all have their victims.

An analysis of the more every-day features of these accidental CO deaths has been published elsewhere[5] *vide supra* in order to reach the general practitioner who so commonly bears the responsibility of making an early decision, and to urge public interest in prevention methods.

Certain medico-legal aspects of the problem which merit closer study then became apparent, and three of these will be discussed here:

1. *The difficulty of detecting low saturation* CO deaths—especially in the elderly, the drunk and the ill.

2. *The danger,* in assessing signs and symptoms, of misinterpreting the observations made—*of raising suspicion of crime* where none existed.

3. *The canard of HbCO dissociation after death*—and in blood samples *in vitro*—a myth of no substance.

1. Detection of Low Saturation CO Deaths

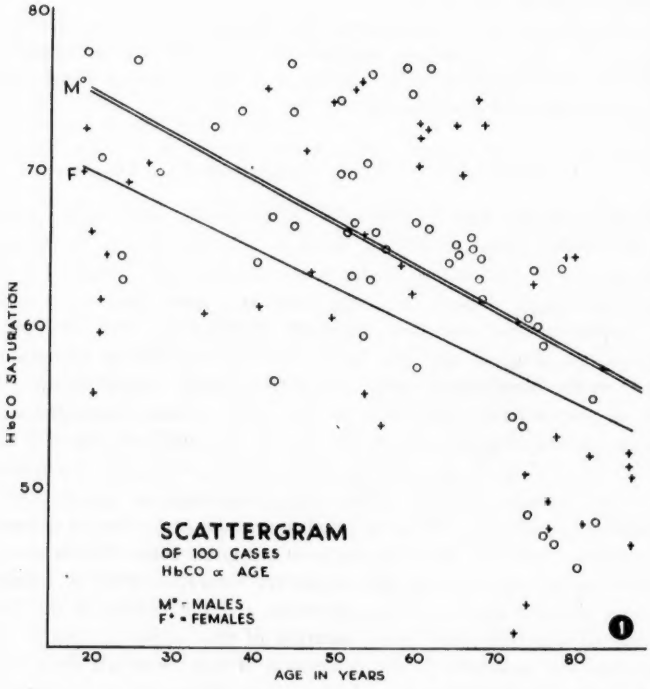
Since the only post-mortem feature of CO poisoning which is not common to many other forms of suboxic death is a colour change in the blood, reflected in the livid stains of the skin—variably well defined and sometimes exceedingly difficult to detect, even in a good light—it is perhaps not surprising that cases are commonly overlooked. The decrepit, the diseased, the drugged and the drunk may all succumb to saturations of HbCO in the blood which cause little colour change, mixed as the HbCO is with preponderant quantities of oxy- and reduced haemoglobin. In anaemic or waterlogged subjects the skin is too pallid to convey a colour to the eye, and in those suffering from disease, drugs or drunkenness a degree of cyanosis may be sufficiently pronounced to obscure any co-existent colour change. Observers' ability to judge red values is notoriously unreliable—commonly non-existent, as in red-green colour-blindness.

There is no general agreement about the saturation level at which the ordinary observer may expect to appreciate a colour change in the blood: indeed, all standard works omit mention of this important point. It is subject to the variability in the perception of reds to which attention has been drawn, but the figure is undoubtedly far below the 30 to 40% level at which serious loss of control (muscular weakness, inco-ordination, diplopia, giddiness and the possibility of collapse and death) ensues in the healthy subject. Haldane's original experiment upon himself noted the blood as being 'distinctly a little pink' at a saturation of 17% HbCO.

The co-existence of any respiratory or circulatory (especially myocardial) deficiency, or of anaemia, results in a significant reduction of the critical minimum lethal saturation: indeed, death is commonly precipitated at saturations in the region of 20 to 30% where no very distinct tint is likely to be present in the blood. Drinkers' well-known monograph on carbon monoxide asphyxia paid considerable attention to this in relation to the maximal permissible concentrations of carbon monoxide in urban and

industrial conditions, showing that, using Henderson's criteria, saturations of 0.06 to 0.07% (i.e. 6 to 7 parts in 10,000) were potentially lethal. As little as 0.01% is, of course, harmful and capable of producing symptoms.

The range in lethal saturation levels as related to 'normal senescence' in the sexes, i.e. without adjuvant disease, is shown by the following scattergram:



Of the series reviewed 49 cases were over 70 years old, 11 suffering from active disease and 7 drunk. These had saturations ranging from 23% to 52%. It plainly behoves the doctor to keep constantly on the *qui vive*—or perhaps it should be *qui mort*—for such eventualities. It is in the interests of public safety that acceleration of death by gas in an elderly or infirm, drunken or otherwise disabled subject should be assessed accurately: it is not merely a matter of dry statistics. The imperative need for autopsy and analysis is amply clear.

Forty-six cases in the series of 100 unselected consecutive cases of accidental CO poisoning remained unrecognized until autopsy—with the

advantage of a better light and exposing blood for test in spite of vigilance it is quite possible for some low saturation deaths to be overlooked even at autopsy.

2. Misinterpretation of Signs and Symptoms

Although it is generally recognized that the sequence of symptoms and the physical signs that develop with rising concentrations of HbCO in the blood are simply manifestations of oxygen lack such as are common to any state of suboxia, their recognition as such is often faulty.

For instance, the states of confusion, muscular weakness, inco-ordination and slurring of speech, nausea, vomiting and collapse are all common features of drunkenness. Any police surgeon must bear in mind the possibility of a defence of inhaling exhaust fumes in a drunk-in-charge. It is more often put forward with the hope of disturbing the confidence of the medical evidence than with the prospect of satisfying the court of innocence. Drunken subjects may, of course, develop further symptoms or meet their death from CO inhalation.

In forensic pathology the 3 features that appear to be most commonly misinterpreted are (a) vomiting (b) livid engorgement, often with petechiae, and (c) cerebral manifestations of suboxia—headache, confusion, giddiness, inco-ordination, loss of consciousness. Vomiting is, of course, a manifestation not of parenteral poisoning but of central nervous system suboxia: it is a late reflex, and taking place during or after loss of consciousness; the vomitus commonly becomes inhaled, precipitating death at a lower saturation and causing cyanosis, pronounced livid staining and sometimes petechiae. That these features should raise suspicion is perhaps not unnatural.

Six people—two adult women and four children—were found in 1954 at Waltham Cross, strewn in considerable disorder around a room of some 10′ x 12′ x 8′ which slept them all. Four were dead, and vomit soiled their lips and nostrils, raising strong suspicion of poisoning by mouth. The husband was suspect for he did not live with the family and stood to benefit (by ceasing allowances) in the event of their death. An empty tin of nicotine was found in the passage. No one—neighbours and relatives, a P.C., a police surgeon and C.I.D. officers called to investigate—had raised the question of fume inhalation, though the bedroom was sealed by a curtained window and door, both shut, and contained a wall gas mantle and a gas fire, still glimmering. There was no ventilation for either, and the atmosphere was appalling. Crime had become strongly suspect because of the circumstances—and the failure to recognize the features of group CO poisoning.

In 1947 a staff waitress of 34 was found dead in a hotel at Reigate. Her husband, who slept with her in a small bedroom, had come down to work late, was confused in both speech and thought, unsteady on his feet, had been unable to lace his boots or make up his bow tie, and was assumed to be recovering from a drunk 'night out'. Search revealed his wife dead in bed with a bedside lamp lying across the head and facial injuries: vomit soiled the lips, chin and neck, and urine had been evacuated. There was no sense of stuffiness in the room, the door having been

open from the time the husband had come down to work, though the window was closed. A small gas fire was turned off.

It was at once suspected that the man had, in a drunken temper, beaten the woman's head in with the table lamp. The injuries were, however, mere skin burns from contact with the hot electric light bulb. The livid stains in the skin were distinctly pink and the blood saturation was 47% (vomit had risen terminally into the glottis). Tests showed a defective gas fire ventilation when the wind was in a certain quarter, fumes accumulating in the room if door and window were shut. It had been a cold night and the suspect husband, who had been drinking, had left the gas fire burning all night. He later developed a large haematoma of the thigh (from muscle necrosis) and a number of suboxic skin blisters—delayed features, together with his symptoms, of sub-lethal CO poisoning.

In cases where the blood has remained fluid or has rapidly become fibrinolysed after death, the livid stains may be pronounced, and where cyanosis is a feature—as in cases lying face down in bedding or on the floor, inhaling vomit or becoming engorged and cyanosed from other causes, the livid stains may be remarkably deep tinted—often shot with petechiae (as may also be the substance of the brain, the neck tissues, heart and lungs). These changes may raise strong suspicion of suffocation or strangulation. Analysis will resolve the doubt that may exist.

A man of 46 was found in a common lodging house dead on his face, hands and knees by the side of his bed. He had arrived home late the previous night with another man and other residents had heard quarrelling. The face was intensely suffused—'almost black' and shot with petechiae: suspicion of foul play arose. Evacuation of faecal and urinary matter was thought to confirm asphyxiation as by throttling. The blood, though cyanotic to the naked eye, showed a 37% saturation by HbCO (facial obstruction having developed upon collapse) and a gas-ring tap was found partly turned on. No smell of gas had been noticed.

Fire may, if it is intense, cause much the same appearance owing to pronounced suboxia accompanying the absorption of CO fumes:

A Scandinavian ship's galley caught fire in the Pool of London, and spread 'with frightening speed' into the adjacent rooms. A draught carried flames into a cabin on the port side where a man sat talking to his wife. He pushed her out but collapsed and died—without a trace of fire injury. Suboxic features were pronounced and the HbCO saturation was 36%. This story was suspect until the significance of extreme suboxia was explained. No other kind of injury was found at autopsy: the victim had not been drinking—and was a perfectly fit man at the time of his death.

3. The Post-Mortem Dissociation of HbCO

It has been plainly established by Gettler and Freimuth[1] that CO does not become absorbed by the dead body: the medico-legal significance of this is too obvious to require elaboration. What is of more substance is the traditional belief that, both in the dead body and in the specimen of blood *in vitro* such rapid dissociation of HbCO takes place that it is important to measure the saturation without delay—to preserve specimens under paraffin, on ice, so as to maintain a saturation comparable with that at death. R. E.

Lane,[3] writing a leader for the *British Medical Journal* this year on *Laboratory Investigations in Suspected Industrial Poisoning*, says:

'Ten millilitres of blood should be collected with a sterile dry syringe and oxalated. The blood should be transferred under paraffin to a test tube and more paraffin should be added to fill the tube which should be securely corked. Shaking should be avoided, the tube wrapped to exclude light, and the specimen taken to the laboratory without delay so that the analysis can be carried out as soon as possible'.

Now this may have some clinical interest, but for the dead it is frank nonsense. Sterility does not exist for the pathologist after death, neither coagulation nor fibrinolysis of the blood matters a jot and time, even repeated exposure to air, effects so little change in the HbCO of the blood specimen that it is quite immaterial whether it is tranferred from a venepuncture under paraffin or taken out of the heart into a test tube.

R. Furbank, my technical assistant, submitted the following graph—one of a number compiled from a month's periodic estimations of the saturation of HbCO of a specimen of blood taken into a 10 c.c. screw-capped sample bottle from a coal-gas suicide, opened, sampled in the reversion spectroscope, closed and again stored, at the laboratory temperatures recorded, without preservative. The fall in the saturation reading is quite remarkably small, provided gross gas-forming decomposition does not supervene.

Of course such stability is not certain to be achieved in all specimens, but such is the firm affinity of CO for Hb that stability is a remarkable feature. It is, indeed, this trait that makes such small proportions of CO in the atmosphere so highly dangerous. It cannot possibly be said that the difference between a saturation of, say 50% and 54%—even reading 60%—is highly significant. In the range below 10% (which is difficult to read spectroscopically) such a difference may have more substance. But in routine medico-legal practice (with figures ranging between 30 and 70%) an error of 5 to 10% matters very little indeed. In industrial medicine, the problem of chronic poisoning by low saturations—between 3 and 15% —constitutes an entirely different problem.

The established medico-legal authorities are in agreement on the slow dissociation of HbCO in the body. Glaister[2] remarks: 'After death carbon monoxide disappears from the blood very slowly and may be detected after long intervals. It has been found 210 days after death . . .'; and most of us will have had similar experiences with the mummified or even decomposed corpse. No CO is formed by gaseous decomposition.

In the case of a decomposed body found some 22 days after death from suicidal exposure to coal gas, the colour change in the blood was so pronounced as to be quite unmistakable, and a spectroscope reading gave a figure of 58%. Decomposition cannot have lowered the figure more than 10% or 12% in the three weeks available, for the lethal saturation is unlikely to have much exceeded 70%.

A sports groundsman was found dead in his pavilion, exposed to an escape of gas, the day after he had last been seen alive. He had evidently toppled out of a chair which was overturned beside the body. His spectacles were smashed against a split in the eyebrow. A reading of the saturation of HbCO in the dried blood on the

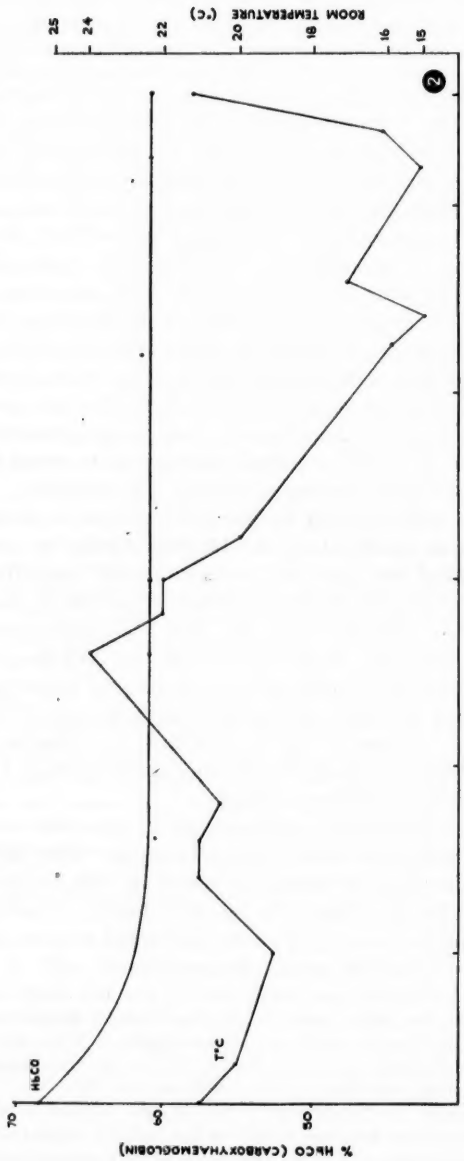


Fig. 1. Graph of HbCO saturation read by Hartridge reversion spectroscope over a period of one month (R. Furbank).

spectacle lens gave a figure of 42%. The blood figure was 68%. It was assumed that CO absorption had already reached a figure of some 42% by the time he had collapsed.

An extra-dural haematoma, found at autopsy in a Welsh villager, victim of a fire that had destroyed her cottage and two occupants—but had permitted the escape of the husband in possession of some 50 charred insurance policies, gave rise to suspicion of foul play: but it was plainly a post-mortem 'boiling' lesion due to intense heat. A measurement of the CO saturation in the clot gave a reading of 40%—confirming the view that it had developed *after* exposure to fire fumes rich in CO. There was no other injury.

The belief that great technical care is necessary to obtain useful blood specimens for CO readings is plainly quite untenable. Absolute accuracy is immaterial to the problem in hand, and a delay of days in the dead body—short of dissolution by putrefaction—is of little matter. *In vitro* the specimen, though, no doubt, better kept cold, can be treated with quite ordinary care, stoppered for security rather than accuracy, and read at convenience.

References

1. Gettler, A. O. and Freimouth, H. C. (1940): Amer. J. Clin, Path., **10**, 603.
2. Glaister, J. (1950): *Medical Jurisprudence and Toxicology.* Edinburgh: E. & S. Livingstone, Ltd.
3. Lane, R. E. (1954): Brit. Med. J., **1**, 978.
4. Trichter, J. and Helpern, M. (1951): Ann. Rep. Amer. Publ. Hlth. Assoc.
5. Simpson, K. (1954): Brit. Med. J., 2, 774.

How Trustworthy is the Witness?

Henry A. Davidson, M.D.*

Cedar Grove, New Jersey, U.S.A.

Spoken testimony is usually the pivot on which a trial turns. To a large extent, in personal injury actions, and almost totally in criminal trials, oral testimony is the basic fabric of the evidence. Considering the tremendous importance of spoken testimony, it is astonishing that so little attention has been paid to methods of evaluating the trustworthiness of evidence. I do not refer here to anything as simple as perjury. The problem is usually not one of deciding whether the witness is deliberately telling a lie. Generally speaking, the witness tells the truth as he sees it. The vital question is: how does he see it?

The psychologist has already made a contribution here. The psychiatrist can make an even greater contribution in the future, provided some mechanism is established for getting the psychiatrist's opinion into the court room.

In numerous experimental situations the psychologist has shown that, even with the most honourable intentions in the world, people do not actually see what is happening and do not recall it with exactness. The role of the psychologist here is largely with normal witnesses. The psychiatrist, on the other hand, has something to say about mentally abnormal witnesses; and where the psychologist has made an essentially experimental approach, the psychiatrist can make a clinical approach.

The whole idea of submitting a *witness* (rather than a party) to a psychiatric examination is a novel one. Courts are not ready to accept the idea, and no instrument now exists for compelling a collateral witness to submit to such an examination. Yet it is becoming increasingly apparent that a malicious witness, a confused one, an emotionally unstable witness or a mentally defective one, can wreak a vast amount of mischief. Surely

* This address was delivered on 8 June 1954 to the Harvard Associates in Police Science, then in convention, in Richmond, Virginia, U.S.A. Dr. Davidson is Chairman of the Psychiatry Section of the American Academy of Forensic Sciences and Assistant Superintendent of the Overbrook Hospital, Cedar Grove, New Jersey, in the United States. He is the author of the now standard *Forensic Psychiatry*, published in 1952 by the Ronald Press, New York City.

modern jurisprudence is equal to the challenge of finding some way of evaluating the mental competency of a witness.

The problem is not one of insane witnesses. A witness who is frankly psychotic presents little of a problem. The psychiatrist today does not have many insane patients. Modern psychiatry works largely with neurotics, addicted, alcoholic, defective or psychopathic persons rather than with psychotic (i.e. insane) ones. If one considers the entire spectrum of emotional illness rather than simply the insane, the psychiatrist's field will embrace a great many fairly common emotional disorders and insanity will constitute but a small part of this.

What are these emotional disorders? The commonest ones, apart from the insanities, are psychoneuroses, psychopathic personalities, mental defectives, alcoholics, drug addicts and persons with organic brain disease. Let us review these and see what these disorders mean in terms of testimonial capacity.

The Psychoses (Insanities)

Frankly insane witnesses seldom appear except in hearings held in mental hospitals. Such hearings may be held when an attendant is charged with cruelty, or in connexion with a writ to get a patient out of the hospital. Occasionally a senile or manic or paranoid psychotic does get into the ordinary courts as an ordinary witness.

The testimony of a senile patient may be tainted by one of these three characteristics: delusions of ingratitude; impairment of memory; delusions of infidelity. One of the common features of senile psychosis is a delusion that the children are cruel and ungrateful, or that the wife (herself, perhaps, an 80-year-old lady) is unfaithful! The memory defect of a senile person is idiomatic. He remembers remote events with clarity, but is foggy and often completely wrong in his memory of recent events. In general, the memory of a senile person is not trustworthy, and it would not seem just to make a judgment if the judgment turns on a senile memory.

Another type of psychotic who occasionally mounts the witness stand is the 'manic'. The word is 'manic', not 'maniac'. A manic is a person who is overtalkative, overconfident, overhappy, overactive. He is very positive in giving testimony, and his psychosis makes him more positive than the facts warrant. Superficially he gives the impression of sanity and stability; but if he is allowed to talk for a while, he will expose himself by reason of his blissful overconfidence, unjustified elation or extreme overactivity. He is a misleading witness.

A paranoid witness seems to make good sense. The word 'paranoid', here refers to a person with a well-organized delusion system (usually a network of persecutory delusions). The paranoid person is suspicious of everyone and everything; he overemphasizes casual occurrences and gives mystic meaning to meaningless incidents. He can make a soft gait sound

like a sneaky one, and he can read into any tone of voice an imaginary nuance of special meaning. His intense suspiciousness makes him an eager witness, but unfortunately, an untrustworthy one. He is so convinced of the justice of the cause he is advancing that he does not mind shading the truth to convince others.

Other psychotic states are less common among ordinary witnesses and, by reason of space limitations, will not here be considered.*

Drug Addiction

There is an impressive amount of law on the credibility of drug addicts. Singer and Krohn [1] cite cases which vary from a ruling that every case must be decided on its merits to a ruling that no drug addict is to be believed. From the clinician's viewpoint, these features of addiction must be remembered. If, apart from the addiction, the witness does not show other evidence of psychiatric abnormality, it is probably safe to assume that, so long as he is receiving his usual dose, he is a reasonably normal person. He cannot be trusted to tell the truth, if that truth might impair the supply of his drug, for that is as precious to him as life itself. But if his testimony concerns something not related to his addiction and if, at the time he witnessed the event, he was not undergoing withdrawal, he should be a reliable witness. On the other hand, the average juror is so disturbed at the conventional picture of the addict (the 'drug fiend' as he is visualized in fiction) that he is uneasy about accepting the testimony of an addict, and lawyers are unwilling to use such testimony if it can be avoided. Scientific objectivity is best served by demanding that any lawyer who wants to impeach the addict's testimony, must prove that for this witness, the addiction did impair his credibility. There is no scientific merit to the automatic disqualification of all addicts.

Mental Deficiency

Mental deficiency is the same as feeble-mindedness,† i.e. it means a basic impairment of intelligence. It must be clearly differentiated from insanity. In general, the distinctions are these: mental defectives are usually born that way; insanity is acquired later in life; mental deficiency is not curable; most insane persons do recover from their psychotic episodes; mental defectives require training, while insane persons require treatment; mental deficiency is essentially an impairment of intelligence. Any other mental faculties are impaired only secondarily. In insanity, on the other hand,

* For a more detailed exposition, see Davidson, Henry A. in *Forensic Psychiatry*, pp. 205-220. New York, 1952. Ronald Press.

† In the United States. In Britain and in many parts of the British Commonwealth, the term 'feeble-mindedness' is reserved for the highest grade of mental deficiency. In the United States, such a person is called a 'moron', while the phrase 'feeble-mindedness' is synonymous with mental deficiency in general.

intelligence is usually normal and may be superior, whereas the other mental faculties are profoundly affected.

Mental defectives are all around us. They engage in humble or menial work, a scarcely noticed backdrop to our activities. They are the invisible men. They are witnesses to all sorts of incidents and often are called to give testimony. The mental defective may be a good witness if the event is one that can be described in a few broad strokes, because he does not embroider much or introduce interpretation. Unhappily, most events are made up of subtle details, and here the defective is a liability because of the poverty of his observing powers and his awkwardness in explaining what he does know. His inability to paint the verbal picture with vividness proves exasperating to the examining attorney and makes him easy prey to the cross examiner.

Alcoholism

If a witness was drunk at the time of an event he is a completely unreliable witness. If he is a deteriorated or psychotic alcoholic, he is likewise untrustworthy. The non-deteriorated but chronic alcoholic is an uncertain witness. The fact that he does not seem deteriorated, gives his words some plausibility. In truth, a chronic alcoholic is an unreliable observer. He is at the mercy of mixed and unpredictable emotions. He is given to periods of mawkish sentimentality, emotional instability and self-pity. There is a probative problem here, too. Courts are loath to receive evidence that a collateral witness is a chronic alcoholic. They usually admit evidence of drug addiction, even though alcoholism can rob a man of his competency more effectively that opium. The reason is the curious paradox, that a drug addict who has received his usual dose of drug is normal (because the dose of drug was needed to neutralize withdrawal symptoms); whereas an alcoholic who has received *his* usual dose of alcohol is certainly not normal.

Psychoneurosis

The word 'psychoneurosis' refers to a set of symptoms due to emotional conflict. Psychoneurosis may mediate itself through physical symptoms. Our concern here, however, is with psychoneurosis manifested by anxiety, depression or fantasy. A child or repressed adult woman, e.g. may develop sexual fantasies and make charges of rape, seduction, indecent exposure and the like, without any basis at all. Orenstein [2] lists cases in which neurotics have acted out unconscious drives and given sincere but completely false testimony leading to the conviction of the innocent. Borchard [3] cites no less than 65 cases in which innocent persons were convicted, largely through individual or mass hysteria which led to well meant but false testimony. Twenty women were once hanged as witches on the sincere and sworn testimony of a number of hysterical girls.[4] Redlich and Ravitz [5] demonstrated

experimentally that neurotic, guilt-ridden and self-punitive persons could easily be induced to make false confessions implicating both themselves and others. Only lately has there been much interest in the effect of pressures on the neurotic mind and the way in which such pressures can lead to false testimony. This has potentialities as an instrument of terror. It certainly makes one want to think twice before making a judgment based largely on the testimony of a neurotic.

Psychopathic Personality

One of the least understood terms in modern psychiatry is the 'psychopathic personality'. Under various other designations, this term is used to describe a sane person of adequate intelligence who is rebellious, dishonest or nonconformist and who gets into trouble because of this personality quirk. The important thing to remember about the psychopath is that he is sane. The semantic difficulty arises because a 'psychopathic ward' is used to house insane patients, whereas a 'psychopath' is someone who is sane. He is sane, but he cannot keep out of mischief because of a personality oddity. Many psychopaths are intelligent, charming and impressive. They make excellent first impressions, but they do not wear well. The psychopath, it soon becomes clear, is not to be trusted, though at first his glibness and apparent amiability make one want to believe in him. When he gets into trouble, which is often, he is charming about it. He promises to reform and enunciates this promise with such impressive firmness that almost every one believes it.

The diagnosis of psychopathy is not made by examination but by a review of the life history. Examination shows nothing. The life history shows a record of trouble, of shiftlessness, of nomadism, of dishonesty, of nonconformity, of mischief or of some similar trait. When this is the history in a witness of good intelligence and obvious sanity, one has the right to suspect psychopathy.

What makes the psychopath such a terribly dangerous witness is that he is willing to incriminate himself to get a moment of exhibitionist glory in the witness box. The jurist and the juryman are alike puzzled by this, for such malevolence is beyond their ken. 'Why would a man manufacture a story which degrades himself? Therefore his story must be true' is the natural response of the juror. Yet in truth, the psychopath likes to tell lies, enjoys using the witness stand as a vehicle for the expression of hostility, is skilled in making a good impression and enjoys wreaking mischief even at the cost of self-degradation. Hoffinger[6] (who is a lawyer, not a doctor) makes this shrewd observation:

'In spite of his disordered personality, the psychopath may appear normal. His lies are told with more conviction that those of normal people. If his lying is exposed, he is capable of quick adjustment, thereby thoroughly misleading the layman. The psychiatrist, however, *can* separate the pathological liar from the

normal liar by correlating the psychopath's maladaptive behaviour into a diagnostic life pattern.'

The distinction between 'pathologic liars' and 'normal liars' here is interesting. A normal lie is told because the liar expects to get some direct and obvious benefit. It is a 'normal' lie for the little boy to avoid a spanking by denying that he took the cookies. It is a 'normal' lie for a burglar to deny the burglary. It is a 'normal' lie for the motorist to say he was going only 25 miles an hour. These lies are told to get an obvious benefit. However much we deplore them, we can all understand them.

But the pathological lie serves no such obvious purpose. It may, indeed, degrade the liar. It is often easily exposed. We have all seen people who misrepresent themselves as being important or wealthy when they are neither. If they do this in circumstances that lead at once to exposure, the lie is pathological. A 'normal' liar does not tell a lie in the face of immediate exposure. A pathological liar actually gets a kick out of fabricating. He enjoys lies and prefers to avoid the truth. At times he tells a self-incriminating lie rather than report a self-serving truth. The pathological liar is a well-know type of psychopath and obviously a pathologic liar can be a dreadful menace in the court room. When to the psychopathic joy of such lying are added the benefits of exhibitionism, personal aggrandizement and popular acclaim, one can see how tempting it is for the psychopath to mount the witness stand and spin an elaborate web of falsehood, with reckless disregard for any persons who might get caught in that web.

The cross-examining attorney is at a singular disadvantage here. How can he prove that the witness is a psychopath? Examination will not reveal it. The life history is diagnostic, but the life history is hearsay and is usually excluded; and this introduces us to our last topic: the tactics of appraising the witness.

The Tactics of Appraising the Witness

Under present rules, a witness cannot be compelled to submit to a psychiatric examination. The testimony may be the result of hysteria, psychopathy, amnesia or feeble-mindedness. The cross-examiner may be absolutely sure of this. But how can he show this to the jury?

Hoffinger[6] suggests that the attorney have a psychiatrist sit at the counsel table and direct the cross-examination, presumably by whispers relayed to the attorney. In a desperate situation, where otherwise injustice would be done, this might be a necessary procedure; but it is a bad plan. The medical profession frowns on it, and the jury cannot understand how a diagnosis can be made without a personal examination.

Actually, the procedure is defensible. Jurors are always making judgments about credibility and mental state by observing the witness. A psychiatrist is

able to do this even better. Yet there is a serious ethical and tactical bar to getting psychiatric opinion based only on court-room observation.

Even if the psychiatrist is willing, the court may not allow the necessary line of questioning. For instance, to prove a senile psychosis it might be essential to ask the witness if he thinks his wife is faithful or his children cruel. But if the old man is a witness to a taxicab accident, no court is going to allow such questions: they seem irrelevant. A question about what the witness had for breakfast this morning is a perfectly proper one for testing memory for recent events. But only a rare magistrate would permit such a question to a witness.

Logically, there ought to be a mechanism for requiring that a witness submit to psychiatric study whenever his testimony is vital and his competence has been questioned responsibly. It seems strange that this indispensable component in the administration of justice is not subject to reasonable cross-checking.

To give effective testimony a witness needs four traits: he must observe intelligently, remember clearly, speak coherently and be free of any emotional drive to distort the truth. The anlysis of these four traits in any witness is a task for the psychiatrist. The spoken word has become too potent a weapon to be placed in the hands of any unstable, hysterical or malicious person, without some mechanism for measuring his ability to handle that weapon. And here is the challenge of truth-seeking in the middle of this twentieth century.

References

1. Singer, H. D. and Krohn, W. O. (1924): *Insanity and the Law*, p. 347. Philadelphia: Blakiston Co.
2. Orenstein, Leo. (1951): Amer. J. Psychiat., **107**, 684.
3. Borchard, E. M. (1932): *Convicting the Innocent*. New Haven: Yale University Press.
4. *See* Starkey, Marion L. (1950): *The Devil in Massachusetts*. New York: Knopf. This Author gives an interesting and detailed account of the neurotic mechanism.
5. Redlich, F. C., Ravitz, L. J. and Dession, George. (1951): Amer. J. Psychiat., **107**, 586.
6. Hoffinger, John. (1950): Yale Law J., **69**, 1330.

The Use and Abuse of Cross-Examination

In Relation to Expert Testimony: The Second Alger Hiss Trial †

Warren P. Hill, LL.B.*

College of Law, The Ohio State University, Columbus 10, Ohio

I will address myself to the problem of why we cross-examine experts, not as an advocate of the *status quo* who accepts the eternal validity of such a practice, nor as a reformer who sees nothing good in the present institution of adversary jury trial. What I will attempt here is a balanced evaluation of the legal practice of cross-examination of the expert witness as exemplified in a particularly well-known trial, with a view to differentiating the legitimate and illegitimate techniques that have become well-embedded in the legal 'art' and practice. This may not have the result of getting my professional auditors to be willing to joyously submit themselves to what ofttimes proves a gruelling and humiliating ordeal, but such sketchy elucidation of purpose *may* serve the worthwhile end of encouraging understanding, in part at least, of why lawyers continue to act like lawyers when they are confronted on the witness stand with an imposing and learned authority whose only wish is to speak the truth as he sees it. Why should his deliverance not be taken *ex cathedra* by the jury? Why all this petty animosity and imputations *ad hominem* officially sanctioned in a proceeding that should be a dispassionate and scientific inquiry into the existence or non-existence of certain disputed facts? And especially, why do we in the legal profession treat the expert so shabbily when we need him so dearly, and sufficient deterrents already exist to keep him shy of the courtroom?

It is easy to see why court appearance with its attendant threat of vigorous cross-examination by opposing counsel is usually an emotionally disturbing experience for the professional man, be he physician, laboratory technician, theoretical scientist or engineer. First, the skilled witness is not accustomed

† A Paper read at a meeting of the American Academy of Forensic Sciences on 26 February 1954.

* Assistant Professor.

to having his views and objectivity put in question by someone not his scientific peer—there is an obvious ego involvement in his personal formulations and judgments. Self-respect cannot be diminished without a consequent painful sensation. Secondly, the courtroom situation is strange, artificial and seemingly arbitrary, and slightly authoritarian in character. The judge towers over the witness like a citadel of technicality; and lastly, there are obvious semantic barriers, augmented by inhibiting procedural rules, which militate against the establishment of an easy rapport with a lay judge and jury.

The degree of unpleasantness associated with a court appearance is, of course, further dependent on the personality and sophistication of opposing counsel. Although he is out to win his case rather than to do justice in any absolute sense, enlightened self-interest dictates against an impulsive headlong attack without planning or specific purpose in mind. As seasoned trial practitioners have repeatedly admonished, 'more often than not, the cross-examination of a truly skilled or expert witness affords him the opportunity to enlarge upon and emphasize his direct testimony'.[1] Abusive and purely destructive cross-examination may often supply its own corrective. But this assumes, of course, that the honest, thoroughly competent, and well-prepared witness can acquit himself adequately in the forensic rough-and-tumble of the courtroom. Not too many experts have a flair or stomach for the game. The laboratory and clinic not being notably conducive to production of such verbal agility, there may even be a negative correlation between such trait and real professional competence and integrity; and yet even if the expert makes undeservedly a miserable showing, the collective heart of the jury may be compassionate enough to take his side against the bully lawyer.

Only Satan himself can decry the exposure on the witness stand of the mercenarily-motivated 'professional witness' or the pompous fraud, so I will assume in this discussion that the major objection taken to present cross-questioning practices relates to the impairment and distortion produced thereby in the evidence given by the sincere, qualified and not consciously biased expert. If we set up certain norms for the purpose of distinguishing abusive from useful types of cross-examination, they cannot be those of the scientific parley seeking to achieve a calm consensus, but must reflect the realities of the present institution of civil or criminal jury trial. The parties in litigation being held responsible for appointing the experts, it must come as no surprise to learn that they customarily seek out only those whose opinions will favour their claim or defense, with only secondary attention given to the prospective witness's professional qualifications to render such an opinion. So although we will seek to determine whether and to what extent present techniques of expert witness cross-examination are conducive to the ascertainment by the jury of the true facts in dispute, we must accept some compromise in our standards in light of the partisan position in which the experts are perforce placed, and the notoriously low minimual standards

of competency exacted in most areas of *expertise*. Experience on the Continent has shown, I might add, that especially in criminal matters, the element of bias is not totally eliminated by court appointment of so-called neutral examiners.[2]

Without dealing further in generalities, let me descend to the details of a portion of a recent federal criminal trial that became the focus of both national and international attention—viz. the case of the United States *versus* Alger Hiss. What I intend to analyse and interpret here is one controversy within the larger controversy over the guilt or innocence of the defendant. About the latter controversy I have nothing very new or incisive to offer. My subject is the evidence given by the defense psychiatrist, Dr. Carl A. L. Binger, and particularly his treatment at the hands of the capable prosecutor Thomas F. Murphy on cross-examination. I select this particular instance of cross-examining an expert witness because it so well illustrates the uses and abuses of this practice and because it involved the application of so many of the commonly approved techniques which trial attorneys employ. It also highlights the basic anomaly of lay appraisal of expert opinion. In addition, the basic facts of the case are well enough known generally so that I need not here delay critical examination of this section of the second Hiss trial.

Dr. Binger was called by the defense in both trials of the perjury charge. His expert testimony, as offered, was directed to the credibility of the chief prosecution witness, David Whittaker Chambers. In the first trial before District Judge Kaufman which resulted in a hung jury, Binger's evidence was held inadmissible by the court because, in its words, 'the record is sufficiently clear for the jury, using its experience in life, to appraise the testimony of all the witnesses who have appeared in this courtroom'. In the second trial Judge Goddard permitted Binger to testify, explaining 'that the value of psychiatry has been recognized. It is apparent to me that the outcome of this trial is dependent to a great extent upon the testimony of one man—Whittaker Chambers. Evidence of insanity is not merely for the judge on the preliminary question of competency but goes to the jury to affect credibility'.[3] The second trial, as you all know, resulted in a verdict of guilty. This outcome is certainly not conclusive on whether the introduction of the psychiatric testimony was ill-advised or not, as we are unable to fathom the thought processes of the jury. The question of the value of this sort of evidence has been much mooted about in the professional literature since then. Many of the people who are still convinced of the defendant's innocence have deprecated it[4] while many who are otherwise disposed with respect to the merits of the case, are generally in favor of the precedent established by the evidentiary ruling in the second trial.[5] If any alignment can be discerned, pro or con the Binger testimony, it is between lawyers and psychiatrists, the latter holding that the good doctor may have delivered his 'profession a blow from which it may take 50 years

to recover'.[6] Modern lawyers, far from being resistant to scientific discovery, tend to grasp at any straw which they feel might perhaps reduce the dreadful uncertainty of litigation. One dissenting legal voice must, however, be noted—the Earl Jowitt in his book on the Hiss case prides himself on the fact that English trial practice tolerates no such nonsense.[7] In all fairness to Binger's professional colleagues who thought he did the cause and the profession a disservice, it is observed that opposition stemmed not from a reluctance to assist our courts in the evaluation of the reliability of the mentally abnormal witness, but from a skepticism regarding the witness' methods and diagnostic criteria.

In response to hypothetical questions, which took over an hour to recite, both Dr. Binger and psychologist Henry Murray concluded that Chambers was suffering from a condition known as psychopathic personality, 'a disorder of character the distinguishing features of which are amoral and asocial behavior, with a tendency towards making false accusations'. A vast amount of time was expended in the examination and cross-examination of these two witnesses—their evidence occupies nearly 450 pages of the transcript.

Before turning to a discussion of prosecutor Murphy's methods of impeachment, let me briefly touch on the problem of admissibility and weight of this sort of evidence, as an understanding of its medico-legal nature may help us decide whether the conclusions of the behavioral scientists were accorded proper reverence at the trial.

Traditionally, a common law jury is supposed to be able to detect the presence of falsehood by observing the demeanor of the witness on the stand, and by balancing his motives for lying against his motives for truth-telling. Opposing counsel on cross will presumably elicit damaging disclosures of bias or interest, obvious defects in observation, recollection and narration, prior inconsistent assertions, past felony convictions, and possibly bring in community reputation for unveracity. These are extrinsic facts from which the jury will infer a subjective element of credibility or deception. But we can only estimate what is going on in a man's mind by putting ourselves in his position, insofar as this is possible. This is the commonsense way of arriving at some conclusions about someone else's motives, *viz. identification.* The premise here is that the personality dynamics of the witness in question are substantially similar to those of the jury—mutual normality.[8]

But suppose the witness is externally 'different'; that although legally competent in the sense that he is not completely out of touch with reality, he has certain observable and well-recognized symptoms of mental derangement. The jury expects now that the witness will be motivated in some peculiar manner not susceptible to the identification method. The identifying link is consciously broken. Hence, other things being equal, this lay body drawn from the general community, will accord greater weight to the opinion of a psychiatric examiner that such-and-such a mental condition,

which has visible signs, bears tellingly on the degree of probability that the 'fact' witness is telling the truth. The testifying psychopathologist here is illuminating a mental terrain which the average citizen has never glimpsed, and assuming that the expert is technically qualified and free from corruption and interest, the jury will normally accept his word as fact in their deliberations. The triers of fact must so accept it, for by hypothesis, they themselves lack the capacity to appraise the testimony effectively.

Now let us consider the witness with no manifest symptoms of mental disorder, who can testify in a rational and coherent manner and possesses intellectual attributes well above the average. No one contends that the man is psychotic or in lawyer's parlance 'insane' for any purpose, but opposing counsel do, let us say, contend that he is a pathological liar due to a behavior disorder known medically as 'psychopathy'. According to their behavior experts this disorder, if present, may impair social judgment and responsibility to a greater degree than paranoia or other functional mental diseases. The judge is faced initially with two problems:

i. Does such a condition exist—has it been medically substantiated? and

ii. Has sufficient observation and study of the alleged psychopath been made so as to afford reasonable certainty that the diagnostic label fits?

On the first point the judge might hold after perusing the relevant psychiatric treatises, that the subject matter, i.e. witness credibility, is such that it can be adequately handled by sound lay intelligence, assertion of the possibility of superior expert handling being a delusion or an empty pretense. To another judge, this clinical entity and its meaning in relation to behavior, may have moved from the precincts of speculation into the realm where *experto credite* holds sway.[9] Logically, because the present medical learning on psychopathic behavior is so far outside common knowledge, the jury can less indulge its common sense about human behavior and is more dependent on expert help in drawing inferences from established data. Of course, we know that the very opposite effect occurs largely because of the common man's emotional resistance to psychiatry. What bolsters this popular antipathy to revelation of the mysteries of emotional health, is the disagreement in this area even amongst the medical workers themselves. Dr. Binger recognized this fact in his book *More About Psychiatry* (1949):

> 'Much of what medical men say and write about is controversial and is overlaid with strong feeling. This is inevitable in a living science. Disagreement exists chiefly in the frontiers of knowledge. Where methods are empirical, where experiment and predictability are not yet possible, where scientific fact is unsupported by adequate theory—there will be differences of opinion and disputation . . . And especially is this true in psychiatry, where methods are less precise, statistics hard to come by, theoretical conceptions tentative. We disagree among ourselves—and hotly, too—on such matters as psychoanalysis, shock therapy, and others.'

Even Dr. Hervey Cleckley who has been in the vanguard in recognizing

the psychopath as a profoundly disordered individual,[10] admits that he (Cleckley) 'is not reluctant to grant that this disorder, viewed theoretically, is a subtle one, and the psychopathology I suggest is debatable and scarcely to be proved in courts.'[11] Psychiatrist Philip Roche has argued that a diagnosis such as Binger made of Chambers is little better than 'name-calling' based as it necessarily was (in view of the socio-pathic nature of the disorder) on value judgment about social behavior.[12] It is a character defect, not an abnormality of personality. Maybe some day if psychiatry continues to develop at its present rate, the matter of psychopathy will enter the realm of general community knowledge, so that even lay witnesses can testify reliably about it (as they can now about 'insanity').

But if the trial judge decides that sufficient medical certainty exists concerning the meaning of the diagnostic term 'psychopathic personality', he will still have to decide whether only clinical observation of the witness whose credibility is challenged will suffice as a factual basis for a competent opinion of this sort. Whether a courtroom diagnosis based on data presented in court is feasible should probably be left for decision to the qualified psychiatrist. At least he is better qualified than the jury to assess personality disorders. Ideally in the Hiss case, the court should have been empowered to order clinical examination of the witness by a court-appointed psychiatrist. Lacking this opportunity to interview and examine Chambers, Binger could only express his opinion in response to a hypothetical question reciting every incident, discreditable or considered suggestive of abnormality, developed in the defense attorney's long cross-examination of Chambers and the testimony of an old school-friend; and based on his study of Chamber's various writings and translations and his observations of him on the witness stand.*

Ideally again, there was no reason why the defendant should have been entitled to a highly selective summation of the most damaging parts of Chamber's testimony and the odder aspects of his behavior—under section 9 of the Model Expert Testimony Act which attempts to remedy this abuse, Binger would have stated his opinion as formulated from his attendance upon the testimony given, but could be required on direct or cross-examination 'to specify the data on which his inferences were based'. After answering the hypothetical in the manner previously indicated, Dr. Binger for a whole day drew telling analogies between these symptoms and Chamber's known behavior and writings. Then began the jousting match with the prosecutor. According to the account of the trial by Francis X. Busch, Murphy 'rose to the occasion magnificently'.[13] Whether this handsome encomium is justly deserved we will now attempt to determine by an

* Binger diagnosed Chambers as a psychopath largely on the basis of 12 characteristics: 1. Repetitive lying. 2. Stealing. 3. Withholding truth. 4. Insensitivity to the feelings of others. 5. Play-acting and assuming false names. 6. Bizarre and unusual acts. 7. Vagabondage. 8. Instability of attachment. 9. Panhandling. 10. Abnormal emotionality. 11. Paranoid thinking. 12. Pathological accusations.

analysis of the measures he attempted to use for depreciation of the direct testimony.

Binger from all accounts made a fairly effective witness for the defense. He was poised and confident, and articulate if not glib. He had had a brilliant career in medicine and was obviously a man of great ability. A shrewd cross-examiner might have approached the challenge of his testimony in generally two quite different ways. The first way, which was *not* followed, would have been to assume a sympathetic and receptive attitude toward the field of knowledge involved, with some display of respect for and grasp of the field. The expert can then often deal with the lawyer in his own terms which can prove dangerous if the latter is only superficially grounded in the subject matter. However, the well-prepared lawyer can often by so doing demonstrate to the jury that the expert has exaggerated his qualifications or has had little experience with the type of case involved. Murphy might have shown, for instance, that Binger's major training, researches and his chief experience had been in the field of internal medicine, and that his knowledge of behavior disorders was somewhat shaky. While this might have been the indicated approach in some accepted technological field, in psychiatry it plainly was not. The jury might perhaps have been impressed by a display of erudition on the part of the prosecutor, utilized skilfully to expose the speculative characater of the science of psychiatry as it relates to diagnosis and treatment of so-called 'borderline' states, but what would be more emotionally satisfying to them would be a debunking operation which tended to pander to their prejudices.

Murphy, of course, may in all sincerity personally reject the findings and insights of modern dynamic psychiatry. In any event, in the words of Alistair Cooke, Mr. Murphy 'asked no title more glorious than that of representing the humble layman. And he stood for the ordeal like Hamlet's Horatio, the time-honoured *punctum indifferens,* more commonly recognized in this country as the gruff, genial, no-nonsense, all-American regular fellow'.[14] Quite a few of the prosecutor's more sarcastic sallies can only be attributed to a distaste for the psychiatric profession in general, e.g., 'Have you been psychoanalysed?' And after Binger had alluded to certain 'extraordinary analogies between the Hiss-Chambers relationship and one between two fictional characters in a German novel translated by Chambers, Murphy interjected rhetorically: 'Did you find any such analogies in the child's book *Bambi?*' (which Chambers had also translated).

But certainly a large number of the questions put by the government man to the psychiatrist were legitimate in eliciting valuable responses going to the weight to be accorded the expert opinion evidence, and would have been proper in any inquiry no matter how dispassionate and fair-minded. Take the matter of interest which the doctor might have in the outcome of the litigation. He was not being paid any fee, nor would he accept one if offered. His wife was associated in some educational work with Priscilla

Hiss. It is hard to say as an abstract matter which kind of expert might show a greater want of scientific objectivity in his testimony—the selfless partisan of what appeared to be a great liberal cause, or the courthouse medical mercenary who works on a contingent fee basis.

Because the hypothetical questions contained only those factual assumptions favorable to the theory of the defense, it was certainly fair game to supplement the hypothesis with additional facts, and ask for an opinion on the hypothesis as modified. For example, Murphy showed that though the doctor had read assiduously in the works of Chambers he was unfamiliar with his more celebrated 'cover pieces' done for *Time* magazine. Binger had based his diagnosis largely on the basis of 12 characteristics, such as repetitive lying, panhandling, instability of attachments. When pressed the doctor could only account for about 'ten lies over a period of 33 years'.* There was absolutely no evidence in the case that Chambers had cadged money on the public streets—another symptom excused. Chambers had been married to his present wife Helen for 18 years—hardly consistent with an inability to form stable attachments. The expert had also made some errors in observations such as the finding that Chambers rarely answered questions directly but qualified his responses with phrases like 'it would have been', or 'it should have been'. Actually, by count in the record, Chambers had used one or other of those expressions only 10 times in 770 pages of the official transcript, whereas defendant Hiss had used them 158 times in 550 pages of testimony.

If any exception could be taken to Murphy's techniques on the score of purposeful distortion, it would be to his method of attacking Binger's conclusion as unjustified by the facts assumed. The technique here is to minimize the importance of each behavioral peculiarity which was allegedly symptomatic of psychopathy. To no apparent avail did the witness protest that he had to consider the totality of the picture. 'Judgment cannot be isolated according to specific parcels of information'. Yet the impression which the prosecutor was attempting to create in the jury's mind was that if each factor standing alone is of no signficance clinically, how can the sum of a series of zeros rise to anything more appreciable. This method of attack is especially devastating and correspondingly unfair in connexion with a diagnosis of psychopathy, which concerns pre-eminently the whole life pattern or style of the individual and does not derive support from the finding of any gross psychoneurotic symptoms.[15]

I certainly do not wish to judge of the wisdom of Dr. Binger's offering himself as expert in this type of case. As students of legal medicine, the question we must seek to answer is whether Binger only slept in the bed he had prepared for himself, or whether current legal rules and practices are so diabolically contrived as to make all such experts look like fools in

* After exacting this admission from the doctor, Murphy then queried: 'What's par for the course, doctor?'

the public gaze. If I prefer to believe the former, it is not because I think the whole psychiatric project ill-conceived, but because in my view, the psychiatrist must take his risks of popular ridicule if the important job of enlightening the public concerning the 'new psychology' is to be carried on. Psychiatry, if it is to make any strides in mental hygiene education must emerge from the clinic and analytic chamber and submit itself to public cross-examination. The verbal tilt between Murphy and Binger, on which was focussed both national and international attention, clearly personified this great issue of our day. While a few such skirmishes may be lost, by such efforts the war may yet be won in terms of eventual wide-spread understanding of the dynamics of human behavior.

Summary

Even though the Government called no psychiatrists of their own to provide diametrically opposed conclusions about the structure and dynamics of Chamber's personality, the problem of the 'battle of experts' was still involved in the second Hiss trial. Pitted against Dr. Binger's tentative findings was the 'four-square common sense' of the prosecutor. There is an obvious anomaly in permitting a lay jury to resolve the conflict, as expert opinion is only admitted when the triers of fact are confronted with issues which cannot be determined intelligently on the basis of ordinary judgment and practical experience gained through the usual affairs of life. Almost a half century ago, Learned Hand suggested in the course of a brilliant article,[16] that the only way out of this paradox was to establish an expert tribunal which would hear the competing theories, reject the spurious or achieve a synthesis of views, and then so advise the trial judge. These findings would presumably then be controlling on the jury.

Of the other techniques of cross-examination of an expert high-lighted by the Hiss trial, many are reasonably defensible as aids to investigation. These would include technical attacks upon training and special competency, and upon personal interest or bias. An especially valuable purpose often pursued by the cross-examiner of the expert witness who is personally immune from impeachment, is the disclosure of additional but not necessarily inconsistent medical data or propositions favorable to the opponent's theory of the case. Here the cross-examiner really makes the witness his own client's expert. Concedely there are absurdities countenanced in the type of cross-questioning consequent upon use of the so-called 'hypothetical question', but the indicated reform here is obviously complete abolition of 'this monstrosity created by the legal imagination'.[17] Frequent complaint is also made of the question-and-answer method of eliciting information from a trained observer. Guttmacher found by means of a questionnaire that many psychiatrists winced at being required to reply to the questions which were given them by answering yes or no, rather than being permitted

to freely express their knowledge and opinions.[18] In justification, the lawyer might argue that only thus can a truly responsive and legally competent expression be secured; and further, that no qualifying information is permanently suppressed as the doctor can signal his distress to proponent's attorney so that the total picture can be developed on re-direct examination. And *mirabile dictu,* Frederic Wertham thinks 'it is a godsend that there are at least some situations where psychiatrists are forced to give a simple answer to a simple question'.[19]

References

1. Busch, F. X. (1949): *Law and Practice in Jury Trials,* §396, p. 637.
2. Ploscowe (1935): *The Expert Witness in Criminal Cases in France, Germany, and Italy,* 2 Law and Contemporary Problems 504.
3. United States *v.* Hiss, 88 F. Supp. 599 (S.D.N.Y. 1950).
4. Bendiner (1950): *The Ordeal of Alger Hiss,* Nation, p. 123.
5. *See* 11 Ala. Lawyer 212 (1950); 30 Neb. L. Rev. 513 (1951); 1950 Annual Survey of American Law 804, 804-07 (N.Y.U. 1951); 59 Yale L. J. 1324 (1950).
6. Quotation from Ellen Johns in the *New Leader,* 14 January, 1950. See also the disapproving view expressed in Guttmacher & Weihofen, Psychiatry and the Law (1952) c. 15, p. 364.
7. Jowitt (1953): *The Strange Case of Alger Hiss,* c. XXI, p. 221.
8. *Cf.* Alexander, F. (1948): *The Fundamentals of Psychoanalysis,* c. I.
9. *See* Ladd, M. (1952): *Expert Testimony,* 5 Vand. L. Rev. 414, 417-21.
10. Cleckley (1950): *The Mask of Sanity,* 2d ed.
11. Ibid, *The Psychopath Viewed Practically,* in *Handbook of Correctional Psychology* (Lindner & Seliger eds. 1947) p. 412.
12. Phillip Roche (1951): *Truth Telling, Psychiatric Expert Testimony and the Impeachment of Witnesses,* 22 Pa. Bar. Ass'n. Q. 140.
13. Busch (1952): *Guilty or Not Guilty?* p. 275.
14. Cooke (1950): *A Generation on Trial* p. 306.
15. Lindner, R. (1944): *Rebel Without a Cause* c. I.
16. Hand, L. (1901): *Historical and Practical Considerations Regarding Expert Testimony* 15 Harv. L. Rev. 40.
17. *See* White, W. A. (1923): *Insanity and the Criminal Law,* p. 86.
18. Guttmacher, M. S. (1953): *The Doctor in Court—Viewpoint of the Psychiatrist,* XIII Maryland L. Rev. 305, 307.
19. Wertham, F. (1953): *A Psychiatrist Looks at "Psychiatry and the Law,"* 3 Buffalo L. Rev. 41, 48.

Subpleural, Subpericardial and Subendocardial Haemorrhages

A Study of Their Incidence at Necropsy and of the Spontaneous Development, after Death, of Subpericardial Petechiae†

I. Gordon, M.B., Ch.B.*
Durban, South Africa
and
R. A. Mansfield, B.Sc. (Chem.), B.Sc. (Bact.)
Durban, South Africa
With the assistance of
J. E. Duncan Taylor, M.B., Ch.B., D.C.P., K. A. B. Kohler, M.B., Ch.B., I. Prinsloo, Arts (Groningen), I. Robertson, M.B., Ch.B., *and* L. G. von Thomann, M.B., Ch.B.

The original purpose of the study described in this paper was to determine whether haemorrhages found in the serous membranes varied in their incidence or in respect of their number, size, colour or distribution in different types of death. In the course of our investigations, however, we found that petechial haemorrhages can develop spontaneously in the serous membranes *after death*. The significance of this observation in relation to any interpretation which might be placed on the diagnostic value of a necropsy finding of petechial haemorrhages in the visceral pleura and the visceral pericardium is considered in this paper.

The Incidence of Subpleural, Subpericardial and Subendocardial Haemorrhages

Material, Method and Results of Investigation

The incidence of subpleural, subpericardial and subendocardial haemorrhages was recorded at 891 necropsies. The post-mortem examinations were

† This work was undertaken with the aid of a grant from the South African Council for Scientific and Industrial Research.
* Professor of Pathology, University of Natal. Formerly Senior Government Pathologist, Union Health Department, Durban, South Africa.

carried out on persons whose deaths had been notified to the Magistrate, Durban, either because it was known or was suspected that the deaths were due to non-natural causes, or because the deceased persons had not been attended by medical practitioners before their deaths. In most of the latter instances the deaths were due to natural causes. In many of these cases the deaths occurred so rapidly that a medical practitioner could not be summoned in time. In other cases no medical practitioner had been called into attendance although the deceased persons had been ill for a period of time. The cases examined were an unselected consecutive series—the only cases excluded from the study were those in which changes of decomposition had developed in the tissues. The distribution of cases in the series of 891 necropsies is set out in Table 1 (Appendix).

In all cases the visceral pleura and the visceral pericardium were examined *in situ* (i.e. before the removal of the lungs and the heart from the thoracic cavity). The incidence, number, size, colour and distribution of the haemorrhages were charted on the record card illustrated in Fig. 1. In each case the age group, sex and race† of the deceased was recorded and the cause of death was charted in accordance with the code classification set out in the *Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death* adopted by the World Health Organization in 1948.[1]

The incidence of subpleural, subpericardial and subendocardial haemorrhages in different causes of death, grouped under the sections set out in the International Classification, is given in Table 2 (Appendix). This table shows that subpleural, subpericardial and subendocardial petechiae are found at necropsy in many different forms of death.

The incidence of the haemorrhages in the various age groups, sex and race groups, is shown in Table 3 (Appendix). Certain differences were found and the significance of these differences was first calculated in respect of racial incidence.*

Racial Incidence. It was found that there was no significant difference in the incidence of subpleural and subpericardial haemorrhages in the different racial groups. In the case of the subendocardial haemorrhages, however, there was a significant difference in the incidence of the haemorrhages between the European and Asiatic groups, with the Asiatic group showing a greater incidence. The higher proportion of deaths from non-natural causes in the Asiatic group might have accounted for this difference. Similar trends were observed on analysis of the racial incidence in adults only. Although there were no significant differences in the incidence of

† The term 'race' has been used to distinguish the four main ethnic groups in South Africa, viz. persons of European descent, Africans (or Bantu persons), persons of Asiatic descent (mainly from India), and a group referred to as Coloured who have a mixed ancestry, predominantly Euro-African in Natal.

* In all calculations, the limit of significance was taken as $\frac{D}{SE} = 3.0$.

Fig. 1. Record Card. The card was designed to provide for automatic coding so that the data could be examined in a series of tabulations prepared by a machine analysis of the cards. The various figures on the card refer to details of the coding system adopted.

subpleural and subpericardial haemorrhages, the incidence of subendocardial haemorrhages in the European group was significantly lower than that in the African and Asiatic groups. As the percentage of known non-natural causes approximated each other in the European and African groups, the greater incidence of subendocardial haemorrhages in the latter as compared with the European group could not be explained solely on this basis.

Sex Incidence. It was found that in all four racial groups the incidence of subendocardial haemorrhages was higher among males than females, but this difference was not significant. The incidence of subpleural and subpericardial haemorrhages was higher among males than females with the exception of the Asiatic group in which the incidence was higher among females. None of these differences, however, was found to have statistical significance.

Age Incidence. It was decided to confine observations to the African racial group alone, as the number of cases available in this group was sufficient for analysis. It was also possible to analyse the figures in respect of the entire group as it was shown that there were no significant sex differences in the incidence of the haemorrhages in this racial group.

The incidence of subpleural haemorrhages appeared to decrease with age from the neonatal to the juvenile period—the incidence rising again in the adult group. It would appear that this trend was not influenced by the percentage of known non-natural causes. This is shown by the closeness of the percentage incidence of known non-natural causes in the juvenile and adult groups.

The difference in the percentage incidence of subpericardial haemorrhages had no statistical significance.

In respect of the incidence of subendocardial haemorrhages, however, there was a significant increase in the incidence of subendocardial haemorrhages from infancy to adult life. This increase did not appear to be influenced by the percentage incidence of known non-natural causes.

Table 4 (Appendix) shows the incidence of the haemorrhages in the stillborn infants and in the 'non-viable' foetuses examined.

The regional distribution of the haemorrhages in the entire series is set out in Table 5 (Appendix). It was noted that there was a close correspondence of this incidence on both sides of the body.

Our records showed that the number, size and colour of subpleural and subpericardial haemorrhages often varied in different zones within limited regions of the visceral pleura and the visceral pericardium. For this reason it was not practicable to analyse our findings in respect of these factors.

The Spontaneous Development, After Death, of Subpericardial Petechiae

In the course of our study we found that petechial haemorrhages which were not observed at the time of the examination *in situ* were often seen

in the visceral pleura and visceral pericardium after the lungs and the heart had been removed from the thoracic cavity. This observation raised the possibility that petechial haemorrhages could develop spontaneously after death in the serous membranes. In order to investigate this possibility the serous membranes were examined *in situ* at intervals over a continuous period. As it was necessary to mobilize and handle the lungs repeatedly in order to examine the visceral pleura satisfactorily, observations were restricted to the visceral pericardium. The examinations were made on an unselected series of deaths due to non-natural causes and to natural causes.

In each case the following procedure was adopted. The post-mortem interval and the time of commencement of the necropsy were recorded. A primary midline incision was made from the level of the thyroid cartilage to the pubis. The incision was deepened between the xiphoid and the umbilicus and the peritoneal cavity was opened in the line of the primary incision. The coverings of the chest wall were retracted laterally, leaving a clean exposure of the costal cartilages. The skin of the neck was then flayed from the underlying tissues without division of any of the superficial veins of the neck. By division of the rib cartilages in series and disarticulation through the sterno-clavicular joints on each side, the sternum was detached without injury of the underlying vessels. The pericardial sac was opened and the time recorded. The visceral pericardium was then carefully inspected for the presence of petechiae. In some cases petechiae were observed in the visceral pericardium at the time of this initial examination, but in other cases no petechiae were seen. The visceral pericardium was re-examined at 2–5 minute intervals over the succeeding 30–90 minutes. Manipulation of the heart on each occasion was limited to the gentle lifting of the apical portion of the heart upwards and forwards for inspection of the posterior surfaces of the atria and the ventricles.

In a relatively large number of cases it was noted that petechiae developed in the visceral pericardium at a variable period after the initial examination*. They not only appeared in cases where no petechiae were seen at the time of the initial examination, but additional ones appeared in cases where haemorrhages *had been* observed at the initial examination. The method adopted in recording our findings is shown in Table 6 (Appendix), which sets out the protocols in 4 of our cases.

The nature of the phenomenon is illustrated in Figs. 2, 3 and 4. Fig. 5 is a photo-micrograph of a section of one of the haemorrhages shown in Fig. 4.

Groups of petechial haemorrhages which developed spontaneously after death were excised in a number of cases and serial microscopic sections

* In the course of our investigation we gained the impression that the development of the petechiae was influenced to some extent by the time of the year at which the necropsies were undertaken. We were able to demonstrate the phenomena to independent observers with greater regularity during the hot, humid months of the year than during the winter months.

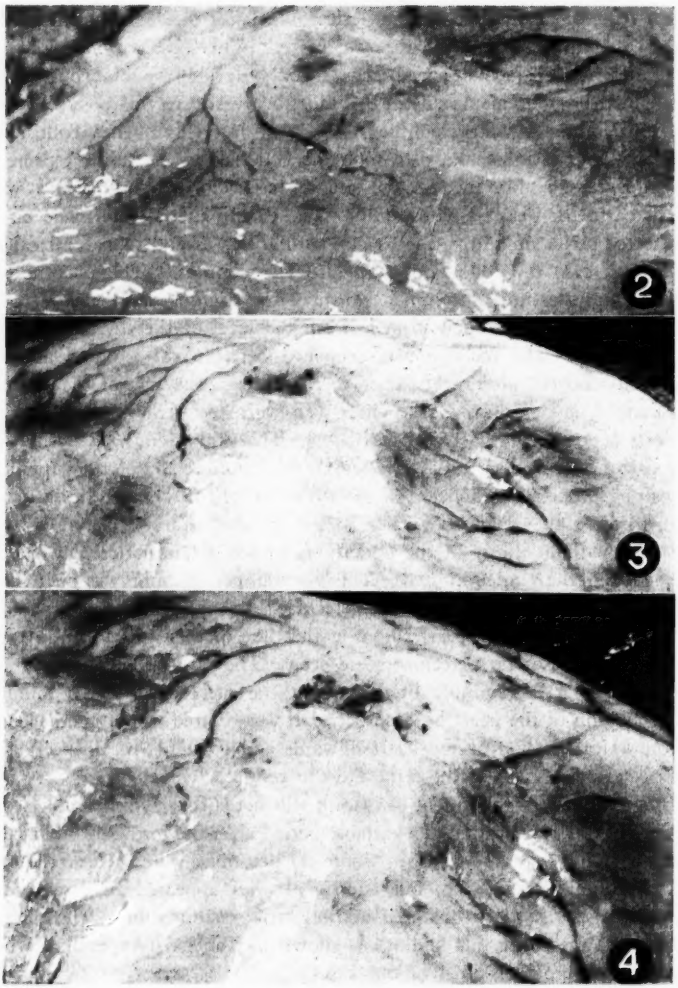


Fig. 2. Deceased—African male adult (aged 48 years). Cause of death—hypertension and arteriosclerosis. Post-mortem interval before commencement of necropsy—4 hours. Pericardial sac opened at 9.52 a.m. Photograph of anterior surface right ventricle taken at 9.54 a.m. Note *absence* of petechial haemorrhages.

Fig. 3. Same case as in Fig. 2. Photograph of anterior surface right ventricle taken at 10.03 a.m. (i.e. 11 minutes after opening of pericardial sac). The photograph was taken from a slightly different angle but the region photographed can easily be identified as the same as in Fig. 2. Note the development of petechial haemorrhages in the pericardium in the zone below the concave bend of the uppermost branching vessel.

Fig. 4. Same case as in Fig. 3. Photograph of anterior surface right ventricle taken at 10.13 a.m. (i.e. 21 minutes after opening of pericardial sac). The photograph was taken from the same angle as Fig. 3. Note the increase in the number of petechial haemorrhages which have appeared in the pericardium.

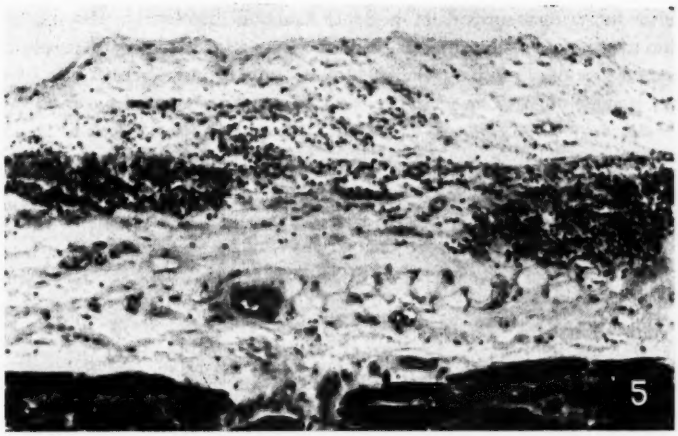


Fig. 5. Photomicrograph of a section of one of the haemorrhages shown in Fig. 4. Iron haematoxylin and eosin (X 150).

were prepared. A control series of serial sections of haemorrhages which were found in the visceral pericardium at the time of the initial examination of the heart was also prepared. These haemorrhages were not distinguishable from each other on histological examination. In both instances extravasations of red cells were found in the connective tissue of the pericardium. Although it was difficult to trace the exact origin or origins of a particular haemorrhage, ruptures of venules were often observed in relation to the haemorrhages. Lacerations or ruptures of arterioles were not seen and there was no clear evidence of capillary ruptures. The general microscopic appearances of petechial haemorrhages which develop spontaneously after death are shown in Figs. 6, 7, 8 and 9. Figs. 7 and 9 are photo-micrographs taken at high power to show the nature of the venular ruptures in these haemorrhages.

Discussion

Petechial haemorrhages in the visceral pleura and visceral pericardium are often referred to as 'Tardieu spots', as they were originally described by Tardieu in a series of papers published during the last century. As Tardieu consistently found these haemorrhages in deaths from mechanical interference with respiration he regarded them as a pathognomonic sign of such deaths.[2] Ogston[3] recorded that soon after Tardieu published his original papers, his claim that petechial haemorrhages in the visceral pleura and pericardium were a specific pathological change was contested by Liman, the celebrated German medico-legal authority. Although the views of Liman

receive substantial support at present, many medico-legal authorities still claim that it is possible to establish that a person has died of 'asphyxia' (in the sense of a mechanical interference with respiration) because of the development of petechiae in the serous membranes, in association with

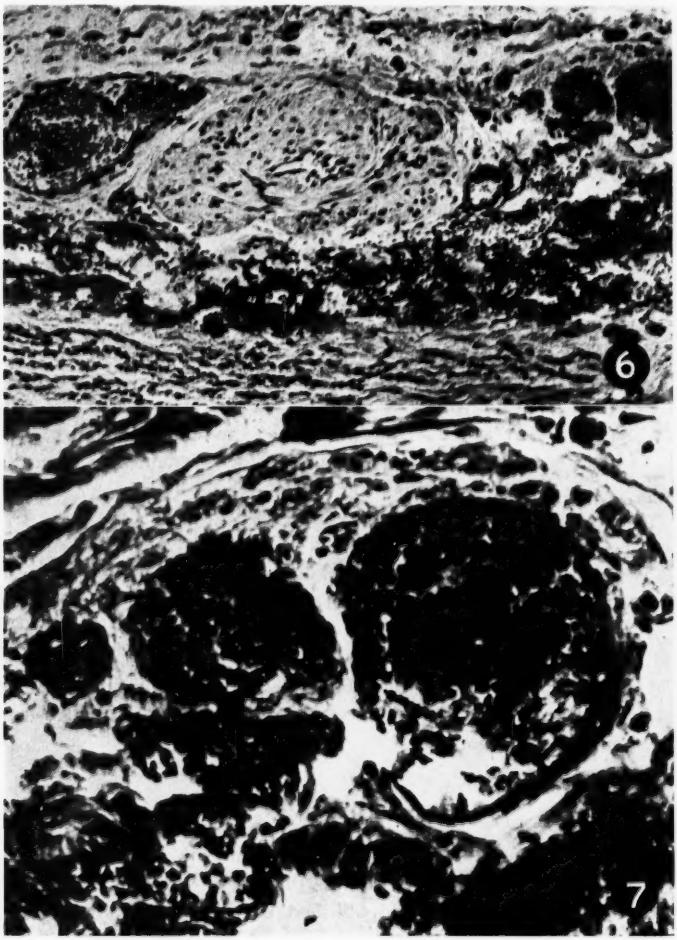


Fig. 6. The general microscopic appearance of a petechial haemorrhage which developed spontaneously in the pericardium after death. Note the engorgement of the venules and the capillaries. A ruptured venule can be seen in the upper right hand zone of the photomicrograph (see Fig. 7). Orthotolidin, haematoxylin and eosin (X 160).

Fig. 7. Photomicrograph of higher power view of ruptured venule seen in Fig. 6. Orthotolidin, haematoxylin and eosin (X 520).

findings such as visceral congestion, cyanosis, cardiac dilatation and post-mortem fluidity of the blood. Gordon and Turner,[4] in an analysis of this problem, suggested that changes such as visceral congestion, cyanosis, cardiac dilatation and post-mortem fluidity of the blood are of a non-specific nature and do not serve to distinguish 'asphyxial' from other forms of death.

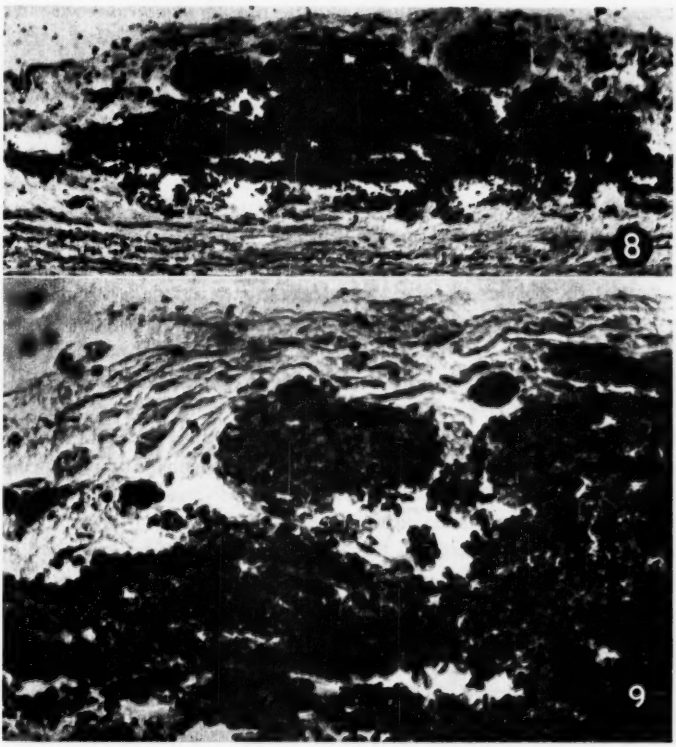


Fig. 8. General microscopic appearance of a petechial haemorrhage which developed spontaneously in the pericardium after death. Note the engorgement of the venules and the capillaries. A ruptured venule can be seen in the upper portion of the section to the left of the mid-line (see Fig. 9). Orthotolidin, haematoxylin and eosin (X 160).

Fig. 9. Photomicrograph of higher power view of ruptured venule seen in Fig. 8. Orthotolidin, haematoxylin and eosin (X 450).

Our present findings demonstrate that petechial haemorrhages may be found in the visceral pleura and pericardium in many different forms of death occurring in persons of various age, sex and race groups. The fact that such haemorrhages can develop *after death* affords the strongest

possible argument against the claim that their occurrence has any pathognomonic significance in the diagnosis of a particular mechanism of death.

It would appear that petechial haemorrhages may develop in the visceral pleura and pericardium before death, at the time of death or after death, but there is no means available of establishing in a particular case, under the ordinary conditions of a necropsy examination, that such haemorrhages were necessarily produced before death or at the time of death and were therefore related to the mechanism of death.

The means whereby petechial haemorrhages develop after death in non-dependent zones of the visceral pericardium has not been established clearly by our findings. The occurrence after death of 'ecchymoses' in the skin within and around zones of lividity in the dependent parts of the body was noted by Haberda in 1898.[5] He described the spontaneous development, after death, of 'ecchymoses' in the skin and, by inference, suggested that when a body is left in the supine position such haemorrhages could also develop over the posterior aspects of the lungs and the posterior aspect of the heart. Apart from the general lividity phenomenon which occurs after death, during which the blood gravitates to all dependent portions of the body, it is possible that there is a zonal lividity phenomenon affecting units of tissue at all levels in a viscus, whereby collections of blood become isolated in tortuous and anastomosing capillaries and venules.‡ The occurrence of post-mortem petechial haemorrhages over the ventral surface of the heart may result from the trapping of blood in a group of dependent venules within a unit of tissue in the ventral pericardium. Such a zonal lividity phenomenon could result in a rise in the hydrostatic pressure within the affected venules and this development might be succeeded by a rupture of the walls of the venules.*

Apart from a possible post-mortem rise in the intravenular pressure during the process of gravitation of the blood into dependent capillaries and venules, as part of a general or zonal lividity phenomenon, there is

‡ In 1951, Shapiro † directed our attention to a case in which petechial haemorrhages, which were observed in the conjunctivae within a few hours after death, had disappeared when the conjunctivae were re-examined 24 hours later. He suggested that it was possible that this phenomenon was due to a post-mortem movement of blood within intact capillaries and venules under the influence of gravity.

We have made two findings which are pertinent to Shapiro's observation. In the first place, in some cases we have observed foci in the visceral pericardium which have resembled petechial haemorrhages on naked-eye examination, but which have proved, on microscopic examination, to be merely collections of blood within dilated capillaries and venules. Secondly, on several occasions we have observed the spontaneous development after death, in the visceral pericardium, of lesions, indistinguishable on naked-eye examination from petechial haemorrhages, which have subsequently faded and, in some cases, have even disappeared altogether.

† Shapiro, H.A. (1951): Personal communication.

* Dissolution of the walls of venules and capillaries occurs during the process of decomposition. There was no evidence of decomposition in the tissues examined in the present series of cases.

evidence to suggest that the intravenular pressure may undergo fluctuations after death, for other reasons. Hooker[6] observed in experiments on animals that there was a movement of blood in the capillaries and venules after death. Within a few minutes of death the blood moves from the arterial to the venous side of the capillary bed and the capillaries and venules become almost completely emptied of blood. About 15 minutes later the capillaries and venules are seen to fill again from the venous side of the capillary bed. After a further period of time (which Hooker claims is coincident with the onset of skeletal muscle rigor), the capillaries and venules are again emptied. This post-mortem movement of blood in the capillaries and venules has also been observed by Carscadden.[7]

Hooker attributes the movement of blood in the vessels after somatic death to the persistence of contractility in the venules, capillaries and arterioles until cellular death supervenes. After cellular death, further contractility may occur with the development of rigor mortis. It is clear that the factors which control the movement of blood in the vessels after death are of a complex nature and a proper understanding of the phenomenon may not be obtained until it becomes possible to measure the pressure gradients within small venules after death.

Regardless of their mechanism of production, it is considered that the mere occurrence of petechial haemorrhages in non-dependent areas of the pericardium and pleura, *after death,* renders invalid the presumption that is often made in medico-legal practice that the presence of so-called 'Tardieu spots' in the serous membranes has pathognomonic significance in the diagnosis of death from mechanical interference with the respiration.

Summary

A study has been made of the incidence of subpleural, subpericardial and subendocardial haemorrhages in a series of 891 necropsies. In all cases the visceral pleura and the visceral pericardium were examined *in situ.*

In each case the incidence, number, size, colour and distribution of the haemorrhages were charted on a card which also recorded the age group, sex and race of the deceased and the cause of death. It was found that subpleural, subpericardial and subendocardial haemorrhages could be demonstrated at necropsy in many different forms of death.

The racial, sex and age incidence of the haemorrhages have been analysed and the close correspondence of the regional distribution of the haemorrhages on both sides of the body was noted. As the number, size and colour of subpleural and subpericardial haemorrhages often varied in different zones within limited regions of the visceral pleura and the visceral pericardium, it was not practicable to analyse the findings in respect of these factors.

In the course of the study it was determined that petechial haemorrhages could develop spontaneously in the pericardium after death. Serial sections of these haemorrhages showed that they were probably caused by ruptures of venules. The mechanism whereby post-mortem petechiae develop in the serous membranes is discussed, both in relation to the occurrence of general and zonal lividity phenomena after death and in relation to the persistence of contractility in the arterioles, venules and capillaries until cellular death supervenes.

The development of petechial haemorrhages in non-dependent areas of the visceral pericardium, *after death*, renders invalid the presumption that is often made in medico-legal practice that the presence of subpericardial and subpleural petechiae has pathognomonic significance in the diagnosis of death from mechanical interference with the respiration.

We wish to thank Mr. S. D. Borsook of Hollerith Machines (S.A.) (Pty.) Ltd. for his assistance in the machine analysis of our record cards, and Mrs. Evelyn S. Bradshaw for her help in the statistical analysis of our data.

References

1. *Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death. Volume 1.* (1949): Bulletin of the World Health Organization Supplement 1. London: H.M. Stationery Office.
2. Tardieu, H. (1866): *A l'Histoire de la Mort par Suffocation*, Ann. d'Hyg. Publ. et de Méd. Légale. **2,** 357.
3. Ogston, F. (1868): *On Punctiform Ecchymoses in the Interior of the Body as a Proof of Death by Suffocation*, Brit. Med. J., **2,** 332.
4. Gordon, I. and Turner, R. (1951): *Deaths from Rapid Anoxia*, Arch. Path., **52,** 160.
5. Haberda, A. (1898): *Ueber das Postmortale Enstehen von Ecchymosen.* Viertaljahreschrift fuer Gerichtliche Medizin, p. 248.
6. Hooker, D. R. (1921): *Evidence of Functional Activity on the Part of the Capillaries and Venules*, Phys. Rev., **1,** 112.
7. Carscadden, W. G. (1927): *Early Inflammatory Reactions in Tissues Following Simple Injury*, Arch. Path. Lab. Med., **4** (3), 339.

Appendix

Table 1: Distribution of Cases in Series of 891 Necropsies

	Number of Cases
Deaths due to natural causes	451
Deaths due to known non-natural causes	368
Cause of death not determined	56
Stillborn infants	14
"Non-viable" foetuses	2
	891

Table 2: The Incidence of Subpleural, Subpericardial and Subendocardial Haemorrhages in Different Causes of Death

(The numbers in parentheses in columns 3 and 4 denote the number of cases in which the presence of adhesions interfered with the recognition of haemorrhages in the visceral pleura and visceral pericardium. In column 3, the first figure in parentheses refers to the number of cases of adhesions in the right pleural cavity and the second figure in parentheses refers to the number of cases of adhesions in the left pleural cavity.)

No.	*Cause of Death*	*Number of Cases*	*Sub-pleural Haemorrhages*	*Subperi-cardial Haemorrhages*	*Subendo-cardial Haemorrhages*
			Infective and Parasitic Diseases		
			(Section I of International Classification)		
1.	Pulmonary Tuberculosis	34	2 (17) (16)	3 (1)	1
2.	Tuberculous Meningitis	3	—	—	—
3.	Tuberculous Peritonitis	1	—	—	—
4.	Tuberculous Pericarditis	1	1	1	—
5.	Disseminated Tuberculosis	7	2 (2) (2)	—	—
6.	Congenital Syphilis	3	1	1	—
7.	Ruptured Aortic Aneurysm (Syphilitic)	11	4 (4) (7)	1	2
8.	Syphilitic Aortitis	29	8 (10) (9)	7 (2)	1
9.	Enteric Fever	4	0 (2) (1)	1	1
10.	Amoebiasis	18	3 (7) (6)	3 (1)	—
11.	Septicaemia	5	2 (2) (2)	2	—
12.	Diphtheria	2	1	1	1
13.	Tetanus	5	—	1	—
14.	Hydatid Disease	1	—	—	—
15.	Blastomycosis	1	0 (0) (1)	—	—
			Neoplasms		
			(Section II of International Classification)		
16.	Carcinoma Stomach	1	—	—	—
17.	Carcinoma Lung	1	0 (1) (1)	—	—
18.	Carcinoma Uterus	1	—	—	—
19.	Carcinoma Testis	1	0 (1) (1)	0 (1)	—
20.	Carcinoma Thyroid	1	—	—	—
21.	Pituitary Adenoma	1	1	1	—
22.	Leukaemia	2	1	2	—
			Allergic, Endocrine System, Metabolic and Nutritional Diseases		
			(Section III of International Classification)		
23.	Nutritional Deficiency	20	3	1	—

Table 2 (*continued*)

No.	*Cause of Death*	*Number of Cases*	*Sub-pleural Haemorrhages*	*Subperi-cardial Haemorrhages*	*Subendo-cardial Haemorrhages*
			Diseases of the Nervous System and Sense Organs		
			(Section VI of International Classification)		
24.	Meningitis (non-Tuberculous)	5	2 (1) (1)	1	—
25.	Intracranial Abscess	3	1 (1) (1)	2	—
26.	Encephalitis	5	3	3	1
27.	Epilepsy (Idiopathic)	1	1	1	—
28.	Otitis Media	22	8	3	—
			Diseases of the Circulatory System		
			(Section VII of International Classification)		
29.	Rheumatic Heart Disease	12	4 (4) (3)	2 (1)	—
30.	Coronary Thrombosis	22	7 (3) (5)	3	1
31.	Ruptured Cardiac Aneurysm following Coronary Thrombosis	2	1 (0) (1)	0 (1)	—
32.	Subacute Endocarditis	1	1	—	—
33.	Acute Myocarditis (non-Rheumatic)	1	—	—	—
34.	Hypertension and/or Arteriosclerosis	49	20 (13) (11)	12	—
35.	Dissecting Aneurysm	3	0 (1) (1)	—	1
36.	Pulmonary Embolism	3	0 (2) (1)	—	—
37.	Subarachnoid Haemorrhage	1	1 (0) (1)	—	—
38.	Cerebral Thrombosis and Haemorrhage	**15**	3 (1) (3)	2	1
			Diseases of the Respiratory System		
			(Section VIII of International Classification)		
39.	Lobar Pneumonia	30	10 (7) (3)	6	—
40.	Bronchopneumonia	23	6 (1) (0)	4	—
41.	Empyaema	2	2 (1) (0)	2	—
42.	Spontaneous Pneumothorax	1	1 (1) (0)	—	—
43.	Lung Abscess	1	0 (0) (1)	—	—
44.	Pulmonary Fibrosis	1	0 (1) (1)	1	1

Table 2 (*continued*)

No.	Cause of Death	Number of Cases	Subpleural Haemorrhages	Subpericardial Haemorrhages	Subendocardial Haemorrhages
			Diseases of the Digestive System		
			(Section IX of International Classification)		
45.	Intestinal Obstruction	5	4 (1) (2)	2	1
46.	Gastro-enteritis	20	4	2	—
47.	Peritonitis	5	0 (2) (2)	—	1
48.	Acute Yellow Atrophy	4	3	2	2
49.	Cirrhosis of Liver	7	2 (1) (1)	—	—
50.	Pancreatic Fibrosis	2	1	1	—
			Diseases of the Genito-Urinary System		
			(Section X of International Classification)		
51.	Nephritis	11	1 (1) (3)	3	—
			Deliveries and Complications of Pregnancy, Childbirth, and the Puerperium		
			(Section XI of International Classification)		
52.	Toxaemia of Pregnancy	1	0 (1) (0)	—	—
53.	Ectopic Pregnancy	2	—	—	2
54.	Abortion with Sepsis	1	1	—	1
55.	Complicated Delivery (Haemorrhage and/or Disproportion)	5	1 (1) (1)	1	2
56.	Puerperal Sepsis	1	—	—	—
			Congenital Malformations		
			(Section XIV of International Classification)		
57.	Congenital Defect Central Nervous System	2	1	1	—
58.	Congenital Heart Disease	2	1	1	—
59.	Congenital Aneurysm—Circle of Willis	2	0 (1) (1)	2	—
			Certain Diseases of Early Infancy		
			(Section XV of International Classification)		
60.	Intracranial Injury at Birth	9	6	4	—
61.	Diarrhoea of Newborn	2	1 (1) (0)	—	1
62.	Haemolytic Disease of Newborn	1	1	1	—
63.	Haemorrhagic Disease of Newborn	5	2	2	1
64.	Prematurity with Incomplete Expansion of Lungs	8	1	2	—
			Ill-Defined Diseases		
			(Section XVI of International Classification)		
65.	Undetermined	56	10 (5) (6)	10	3

Table 2 (*continued*)

No.	*Cause of Death*	*Number of Cases*	*Sub-pleural Haemorrhages*	*Subperi-cardial Haemorrhages*	*Subendo-cardial Haemorrhages*
			Accidents, Poisonings and Violence (Section XVII of International Classification)		
66.	Electrocution	6	5 (2) (0)	3	—
67.	True Anaesthetic Deaths	20	4 (3) (4)	2	2
68.	Fracture Vertebral Column with/without Spinal Cord Lesion	4	2 (1) (1)	1	—
69.	Multiple Injuries	92	26 (22) (16)	18 (1)	38
70.	Intracranial Injuries with/without Skull Fractures	57	21 (16) (15)	25	13
71.	Burns	49	15 (9) (8)	13	12
72.	Ethyl Alcohol Poisoning	2	0 (0) (1)	0 (1)	—
73.	Methyl Alcohol Poisoning	1	—	—	1
74.	Paraffin Poisoning	3	2 (0) (1)	2	—
75.	Turpentine Poisoning	1	1	—	—
76.	Corrosive Poisoning	5	1	1	—
77.	Carbon Monoxide Poisoning	3	2	2	—
78.	Morphine Poisoning	1	0 (1) (0)	1	—
79.	Barbiturate Poisoning	3	2 (1) (0)	1	—
80.	Salicylate Poisoning	3	—	1	—
81.	Sedative Poisons (other than barbiturates)	2	1	—	—
82.	Snake-bite Poisoning	3	1	2	1
83.	Cyanide Poisoning	1	1	1	—
84.	Lightning	1	—	—	—
85.	Exposure	1	1	1	—
86.	Drowning	27	8 (5) (2)	6	2
87.	Suffocation, Strangulation, Hanging	14	10 (2) (5)	7	1
88.	Air embolism	3	1 (2) (2)	1	—
89.	Haemorrhage following Trauma	30	9 (9) (6)	5	10
90.	Sepsis following Trauma or burns	25	13 (4) (4)	8 (1)	3
91.	Anaesthetic Death (Associated Surgical Shock)	4	1 (2) (2)	1	—
92.	Firearm Wound—Head	5	2 (1) (1)	—	1
93.	Firearm Wound—Spinal Cord	1	—	—	—
94.	Firearm Wound—Heart	1	1	—	—
			Stillbirths, Diseases of the Foetus (Supplementary International Classification)		
95.	Stillbirths	14	8	9	—
96.	Non-viable Foetuses	2	1	1	—
	Totals	891	279	213	109

Table 3: The Incidence of Subpleural, Subpericardial, and Subendocardial Haemorrhages in Persons of Different Age Groups, Sex and Race

Age Groups	European					African					Coloured					Asiatic				
	No. of Cases	Subpleural	Subpericardial	Subendocardial	% of Known Non-Natural Causes	No. of Cases	Subpleural	Subpericardial	Subendocardial	% of Known Non-Natural Causes	No. of Cases	Subpleural	Subpericardial	Subendocardial	% of Known Non-Natural Causes	No. of Cases	Subpleural	Subpericardial	Subendocardial	% of Known Non-Natural Causes
Male:																				
Neonatal	4	1	1	—	0·0	22	12	9	1	0·0	2	2	2	—	0·0	3	1	—	—	0·0
Infant	4	3	2	—	25·0	49	11	11	—	8·2	—	—	—	—	—	6	2	—	—	50·0
Child	10	6	6	3	70·0	38	8	12	4	26·3	2	—	—	—	50·0	13	5	4	3	61·5
Juvenile	—	—	—	—	—	7	2	3	2	71·4	—	—	—	—	—	3	1	2	1	66·7
Adult	98	39 39·8%	25 25·5%	7 7·1%	45·9	260	81 31·1%	63 24·2%	47 18·1%	51·5	12	6 50·0%	3 25·0%	4 33·3%	58·3	56	12 21·4%	12 21·4%	14 25·0%	76·8
Totals	116	49 42·2%	34 29·3%	10 8·6%	45·7	376	114 30·3%	98 26·1%	54 14·4%	40·6	16	8 50·0%	5 31·3%	4 25·0%	50·0	81	21 25·9%	18 22·2%	18 22·2%	69·1
Female:																				
Neonatal	2	1	—	—	0·0	18	7	5	1	5·6	1	—	—	—	0·0	3	2	1	—	0·0
Infant	1	1	—	—	0·0	41	12	5	1	9·8	3	—	—	—	0·0	2	—	—	—	0·0
Child	—	—	—	—	—	26	6	2	—	23·1	2	1	1	—	50·0	12	1	2	1	66·7
Juvenile	2	1	1	—	50·0	14	1	2	—	35·7	—	—	—	—	—	10	4	2	3	100·0
Adult	38	10 26·3%	5 13·2%	1 2·6%	36·8	84	21 25·0%	15 17·9%	11 13·1%	34·5	6	2 33·3%	— 0·0%	2 33·3%	83·3	19	7 36·8%	7 36·8%	3 15·8%	57·0
Totals	43	13 30·2%	6 14·0%	1 2·3%	34·9	183	47 25·7%	29 15·8%	13 7·1%	29·5	12	3 25·0%	1 8·3%	2 16·7%	50·0	46	14 30·4%	12 26·1%	7 15·2%	63·0
Male and Female Combined:																				
Neonatal	6	2	1	—	0·0	40	19 47·5%	14 35·0%	2 5·0%	2·5	3	2	2	—	0·0	6	3	1	—	0·0
Infant	5	4	2	—	20·0	90	23 25·6%	16 17·8%	1 1·1%	8·9	3	—	—	—	0·0	8	2	—	—	37·5
Child	10	6	6	3	70·0	64	14 21·9%	14 21·9%	4 6·3%	25·0	4	1	1	—	50·0	25	6	6	4	64·0
Juvenile	2	1	1	—	50·0	21	3 14·3%	5 23·8%	2 9·5%	47·6	—	—	—	—	—	13	5	4	4	92·3
Adults	136	49 36·0%	30 22·1%	8 5·9%	43·4	344	102 29·7%	78 22·7%	58 16·9%	47·4	18	8 44·4%	3 16·7%	6 33·3%	66·7	75	19 25·3%	19 25·3%	17 22·7%	72·0
Totals	159	62 39·0%	40 25·2%	11 6·9%	42·8	559	161 28·8%	127 22·7%	67 12·0%	35·4	28	11 39·3%	6 21·4%	6 21·4%	50·0	127	35 27·6%	30 23·6%	25 19·7%	66·9

The Table does not include 2 cases (1 neonatal female and 1 male infant) in which the race could not be determined. In the case of the neonatal female, haemorrhages were found in the pleura, giving the incidence of subpleural haemorrhages in the entire series (including the stillbirths and non-viable foetuses) at 279 cases (see Table 2).

Table 4: The Incidence of Subpleural, Subpericardial and Subendocardial Haemorrhage, in the Stillborn Infants and Non-Viable Foetuses Examined

Sex and Race	*Number of Cases*	*Subpleural Haemorrhages*	*Subpericardial Haemorrhages*	*Subendocardial Haemorrhages*
Stillborn Infants				
Male African	4	2	2	—
Female African	7	4	5	—
Male Asiatic	1	1	1	—
Female Asiatic	2	1	1	—
Totals	14	8	9	
Non-Viable Foetuses				
Male Asiatic	1	1	1	—
Female (race not determined)	1	—	—	—
Totals	2	1	1	

Table 5: Regional Distribution of Subpericardial, Subpleural and Subendocardial Haemorrhages in Descending Order of Frequency of Occurrence

	Region	*Number of Cases*		*Region*	*Number of Cases*
	L. Vent. Base Post.	86		L. Vertebral	149
	R. Vent. Base Ant.	68		R. Vertebral	139
	R. Vent. Base Post.	54		R. Interseptal	135
	L. Vent. Base Ant.	50		L. Interseptal	123
	L.V.A. Undefined*	45	Subpleural	L. Basal	83
	L. Atrium Post.	36	Haemorrhages	L. Ant.-lateral	80
	L.V.P. Undefined	35		R. Ant.-lateral	70
	R.V.P. Undefined	29		R. Basal	69
	R.V.A. Undefined	27		L. Medial	53
Subpericardial	R. Atrium Ant.	25		R. Medial	51
Haemorrhages	R. Atrium Post.	22			
	L. Atrium Ant.	22			
	R. Infundibular	16			
	L. Aortic	14			
	Inter-sept. Post.	13		L. Septal	99
	Inter-sept. Ant.	9		L. Papillary	60
	L. Vent. Apex Ant.	8	Subendocardial	R. Papillary	5
	L. Vent. Apex Post.	7	Haemorrhages	R. Septal	2
	R. Vent. Apex Ant.	5		R. Atrium	1
	R. Vent. Apex Post.	5		L. Atrium	—

* The term "Undefined" refers to those cases where haemorrhages were found in the visceral pericardium, either anteriorly or posteriorly, in the region between the base and the apex of the ventricle.

L. = left. R. = right. V. or Vent. = ventricle. A. or Ant. = Anterior. P. or Post. = Posterior. Inter-sept. = Interseptal region.

Table 6: Protocols Showing the Spontaneous Development, after Death, of Subpericardial Petechiae

Case	*Time Interval (Minutes)*	*Time*	*Observations*
1. Asiatic Male Adult (aged 32 years).	—	7.50 a.m.	Necropsy commenced.
Cause of death: Acute cyanide poisoning.	0	7.55 a.m.	Pericardial sac opened; 1 petechia seen over anterior surface aorta at its base and 1 petechia seen over posterior surface right ventricle.
Post-mortem interval before commencement of necropsy: 24 hours (body was placed in refrigerator chamber before dissection).	*Additional Petechiae Appeared as Follows*		
	33	8.28 a.m.	1 additional petechia seen over posterior surface right ventricle; 4 petechiae appeared over anterior surface left ventricle.
	35	8.30 a.m.	Localized shower of petechiae appeared in area $\frac{1}{2}'' \times \frac{3}{8}''$ over anterior surface left ventricle.
	50	8.45 a.m.	1 petechia appeared over apical region anterior surface left ventricle; 3 additional petechiae seen over anterior surface aorta at its base.
	55	8.50 a.m.	Localized shower of petechiae appeared in area $\frac{1}{2}'' \times \frac{1}{2}''$ over anterior surface aorta at its base; 1 petechia appeared over posterior surface right atrium.
2. European Male Adult (aged 74 years).	—	8.15 a.m.	Necropsy commenced.
Cause of death: Coronary thrombosis.	0	8.24 a.m.	Pericardial sac opened; no petechiae seen.
	Petechiae Appeared as Follows		
Post-mortem interval before commencement of necropsy: 2¼ hours (body was *not* placed in refrigerator chamber before dissection).	26	8.50 a.m.	Showers of petechiae appeared over posterior surface left ventricle, and over anterior surface left atrium.
	36	9.00 a.m.	1 Petechia appeared along middle lateral border anterior surface right ventricle; 3 petechiae seen in upper portion anterior interseptal region.
	41	9.05 a.m.	Localized shower of petechiae appeared in posterior interseptal region.
3. Asiatic Female Adult (aged 40 years).	—	8.35 a.m.	Necropsy commenced.
Cause of death: Multiple burns.	0	8.43 a.m.	Pericardial sac opened; 1 petechia seen over anterior surface pulmonary artery at its base; small group petechiae (under 10 in number) seen over

Table 6 (*continued*)

Case	*Time Interval (Minutes)*	*Time*	*Observations*
Post-mortem interval before commencement of necropsy: 11½ hours (body was placed in refrigerator chamber before dissection).			posterior surface left ventricle, and 1 petechia seen over posterior surface left atrium.
			Additional Petechiae Appeared as Follows
	3	8.46 a.m.	A slightly darkened area over the anterior surface of the right ventricle, seen at the time of opening the pericardial sac, became much intensified in colour, and a relatively large petechia appeared in this zone of involvement.
	10	8.53 a.m.	In the area over the posterior surface of the left ventricle, described at 8.43 a.m. as showing a group of petechiae (under 10 in number), numerous additional petechiae had appeared. These petechiae were now too numerous to count. 1 Petechia appeared over posterior surface ventricle.
	22	9.05 a.m.	2 Petechiae appeared over anterior surface aorta at its base; 3 petechiae appeared over posterior surface pulmonary artery at its base.
	27	9.10 a.m.	Localized shower of petechiae appeared in area ¼″ × ½″ over posterior surface aorta at its base.
4. African Male Adult (aged 31 years).	—	8.00 a.m.	Necropsy commenced.
Cause of death: Pulmonary tuberculosis.	0	8.08 a.m.	Pericardial sac opened; no petechiae seen.
			Petechiae Appeared as Follows
Post-mortem interval before commencement of necropsy: 1¼ hours (body was *not* placed in refrigerator chamber before dissection).	9	8.17 a.m.	3 Petechiae appeared along middle lateral border anterior surface left ventricle.
	22	8.30 a.m.	1 Petechia seen in posterior interseptal region; 2 additional petechiae appeared along lateral border anterior surface left ventricle; localized shower of minute petechiae seen along course of anterior branch of left coronary artery.
	36	8.44 a.m.	Increase in numbers of petechiae in all regions where they had appeared; petechiae now generalized over anterior surface left ventricle and in posterior interseptal region.

Sex Differences in the Foetal Sciatic Notch

Barbara J. Boucher, B.Sc. (Lond.)
Anatomy Department, London Hospital Medical College

The foetal pelvis, including its cartilaginous portion, has been studied by Thomson,[1] but owing to previous fixation of the tissue little attention was paid to the differences in dimensions which he found between the sexes. The sciatic notch in the foetus has also been examined by Villemin[2] who commented on sex differences to be seen after the fourth month of pregnancy, but he did not express them metrically. (See also Lazorthes and Lhez[3]).

Recently, during the preparation of 5 foetal skeletons from their soft tissue remains, in order to estimate the foetal ages which were required for forensic purposes, the ilia were seen to have either shallow or deep sciatic notches. The two bodies whose ilia had shallow notches were found from the soft tissues to be female. A collection of ilia from stillborn infants or foetuses of known sex was then made, and the dimensions of the sciatic notch were determined to see whether their examination might be of value for forensic purposes.

Methods

Both ilia, together with the left femur, were removed from some 20 foetuses, 6 months to term in age. The soft tissues and cartilage were removed after boiling of the parts. The bones were thus prepared in a state similar to those occasionally obtained from bodies whose soft tissues are useless for the identification of sex. The collection of bones of known sex was numbered so that the sex of each was not known to the writer until after it had been measured.

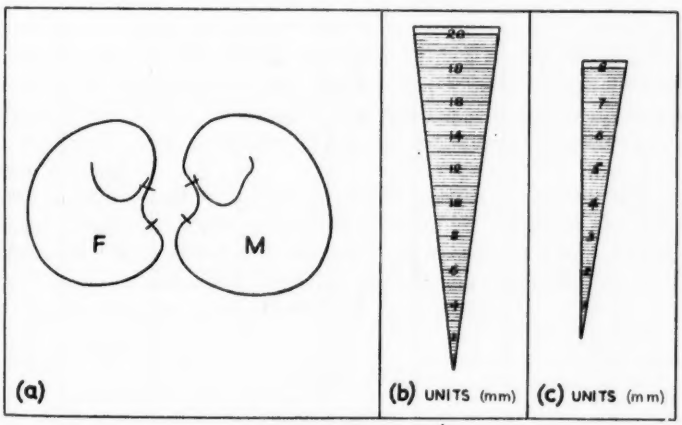


Fig. 1: (a) Line diagrams from an enlarged photograph of male and female ilia, showing their ventral surfaces.
(b) The gauge used to measure width.
(c) That used to measure depth, showing the method of calibration.

Table 1: Width, Depth and Index.

(Recorded for Each Ilium in Order of Femur Length which is Proportional to Age).

	Female			*Male*		
Femur Length (Cm.)	*Width (Mm.)*	*Depth (Mm.)*	*Index*	*Index*	*Width (Mm.)*	*Depth (Mm.)*
2·75				—	—	—
2·75				4·5	3·2	0·7
3·5				3·9	3·1	0·8
3·5				4·3	3·0	0·7
3·6				4·3	3·0	0·7
3·6				4·45	4·0	0·9
3·8	3·5	0·6	5·8			
3·8	3·0	0·6	5·0			
3·8	4·0	0·6	6·63			
3·8	3·0	0·6	5·0			
4·0	4·0	0·7	5·7			
4·0	3·2	0·7	4·6			
4·3				4·65	4·2	0·9
4·3				4·85	4·8	1·0
4·9	4·5	0·8	5·6			
4·9	4·2	0·7	6·0			
4·9	5·8	0·8	7·3			
4·9	5·0	0·8	6·2			
6·1	5·0	0·9	5·5			
6·1	6·4	1·2	5·3			
6·6	8·0	1·46	5·45			
6·6	7·2	1·4	5·1			
6·8	5·8	1·1	5·25			
6·8	—	—	—			
7·0				5·0	6·0	1·2
7·0				4·35	7·0	1·6
7·3				4·2	8·4	2·0
7·3				4·65	7·0	1·5
7·5	9·2	1·6	5·7			
7·5	7·5	1·2	6·25			
7·6				4·1	6·2	1·5
7·6				3·95	7·5	1·9
8·0	7·2	1·1	6·5			
8·0	9·2	1·76	5·25			
8·0	8·6	1·2	7·2			
8·0	7·0	1·1	6·45			

Each ilium was laid on its dorsal surface on a flat table and examined with one eye from about 12 inches above it, and the point of contra-flexure (Fig. 1a) was marked with dilute coloured varnish on each side of the notch. This point is the one at which the curvature of the notch changes to that of the contiguous bone edge, and can be seen by eye. The width between these points was measured by holding the bone with the two marks horizontally aligned against a thin brass gauge calibrated in mm. (Fig. 1b) and held upright from the table. With the bone held firmly in this position with one hand, the depth was measured by running a second scale (Fig. 1c) down the width scale till it fitted snugly between it and the deepest part of the notch. This scale was held so that the mm. lines ran at right angles to the width scale. No force was used in fitting these gauges, and the bones were re-positioned if any shift occurred during measurement. With care consistent results were obtained.

The width divided by the depth of the sciatic notch was used as an index for comparison of the bones. Separate studies of width and depth were not possible because comparison would have had to be made between equivalent age groups, as estimated from the length of the femur, and such groups did not exist in the small samples so far available.

Results

The values obtained are shown in Table 1. No relationship between femur length (or age) and the index is apparent. The statistical analysis used to test the differences between the male and the female indices is shown in Table 2, where it can be seen that these differences should only occur by chance less than once in a hundred such investigations, and are thus statistically significant.

Discussion

The type of difference found between male and female sciatic notches in the adult also occurs in the ossified part of the foetal ilium, and it has been found possible to express this difference mathematically and thereby to subject it to a statistical analysis which it has withstood despite the small sample sizes. Whether the foetal notch varies as it is said to in the adult, in abnormal sexual conditions, cannot yet be stated, but such cases are being collected.

As in anthropometrical work in general, comparison of this index, for sexing a bone, should be made with the recorded indices of the relevant population. It is intended to prepare such records for European infants, but it would be useful and possibly of anthropological and forensic interest to compile tables for Negroid and other racial groups.

Summary

1. A method of obtaining a Sciatic Notch Index for the foetal ilium is described.

2. The Index obtained is found to be significantly larger in the female than in the male in the 30 specimens examined.

Table 2: Showing the Calculation of T, and the Probability of the Results being due to Sampling Error

Index Female X	$X-\overline{X}$	$(X-\overline{X})^2$	*Index* Male Y	$Y-\overline{Y}$	$(Y-\overline{Y})^2$
5·8	0	0	4·5	·1	0·01
5·0	0·8	0·64	3·9	·5	0·25
6·63	0·83	0·69	4·3	·1	0·01
5·0	0·8	0·64	4·3	·1	0·01
5·7	0·1	0·01	4·45	·05	0·003
4·6	1·2	1·44	4·65	·25	0·063
5·6	0·2	0·04	4·85	·45	0·2
6·0	0·2	0·04	5·0	·6	0·36
7·3	1·5	2·25	4·35	·05	0·003
6·2	0·4	0·16	4·2	·2	0·04
5·5	0·3	0·09	4·65	·25	0·063
5·3	0·5	0·25	4·1	·3	0·09
5·1	0·7	0·49	3·95	·45	0·2
5·25	0·55	0·3			
5·7	0·1	0·01			
6·25	0·45	0·2			
6·5	0·7	0·49			
5·25	0·55	0·3			
7·2	1·4	1·96			
6·45	0·65	0·423			
5·45	0·35	0·123			

Average $= \underline{5{\cdot}8} = \overline{X}$ $\quad S = \underline{10{\cdot}55}$ $\quad$ Average $= \underline{4{\cdot}4} = \overline{Y}$ $\quad S = \underline{1{\cdot}302}$

$$\text{S.D.} = \sqrt{\frac{10{\cdot}55}{20}} = 0{\cdot}726 \qquad \text{S.D.} = \sqrt{\frac{1{\cdot}302}{12}} = 0{\cdot}329$$

$$\text{S.E.} = \frac{0{\cdot}726}{\sqrt{21}} = 0{\cdot}158 \qquad \text{S.E.} = \frac{0{\cdot}329}{\sqrt{13}} = 0{\cdot}0916$$

$$T = \frac{\overline{X}-\overline{Y}}{\text{S.E. of the means.}}$$

$$= \frac{1{\cdot}4}{\sqrt{0{\cdot}158^2 + 0{\cdot}0916^2}} = 7{\cdot}68$$

(Degrees of freedom = 32.)

Therefore p = less than 0·01 that the difference is due to chance.

3. The difference between the indices is sufficient to suggest that sex can be determined confidently from the ilium during foetal life.

The writer gratefully thanks Prof. R. J. Harrison for his helpful encouragement, and Dr. F. E. Camps for the provision of the material which suggested this investigation.

References

1. Thomson, A. (1899): J. Anat. Lond., **33**, 359.
2. Villemin, F. (1937): *La Differenciation Sexuelle Precoce de la Grande Echancrure Sciatque, et son Retentissement sur l'evolution du Bassin de l'homme.* Strasbourg Med., **33**, 593.
3. Lazorthes, G. and Lhez, A. (1939): *La Grande Echancrure Sciatique, Etude de sa Morphologie et de ses Caracteres Sexuels.* Arch. d'Anat. et d'Hist. et d'Emb. (Arch. Anat. Strasbourg), **27**, 163.

The Significance of Manganese in Human Tissues

I: The Determination of Manganese in Biological Material

P. R. v. d. R. Copeman, B.A., D.Sc.

Government Chemical Laboratories, Johannesburg

Although cases of poisoning by manganese compounds, especially potassium permanganate, are somewhat unusual, the need of a method for the determination of small amounts of manganese in the tissues is apparent. In addition, since manganese is normally present in human tissues, it is also important to know what amounts can normally be expected in such tissues. The present work was undertaken with these objects in mind.

The usual methods for the determination of manganese in small amounts depend upon its oxidation to permanganate, and the resulting pink solution compared with standards. Various oxidizing agents have been proposed by different workers,[1, 3] and of these, persulphate, bismuthate and periodate have proved the most successful. The periodate method, proposed by Willard and Greathouse,[4] possesses certain advantages which are absent in the other methods. In the first place, it does not require the presence of a catalyst as in the case of the persulphate method and, secondly, the solution after oxidation does not require filtration to remove excess of the reagent as is the case with the bismuthate method. In the present work the periodate method proved entirely satisfactory.

Richards[2] has investigated in some detail the periodate method for biological material. For the determination of small amounts of manganese, two factors in particular require attention, viz. (*a*) the acidity of the solution and (*b*) the removal of chlorides. In general, the danger lies in excess rather than in deficiency of acidity and it is recommended that, in dealing with animal tissues the final acidity should be about 5-6%. Below 1% acidity fading takes place and above 15% the colour of the solution fades to a yellowish tint. The presence of chlorides leads to the formation of chlorine which interferes with the oxidation. In the present work it was noticed that, when chloride was imperfectly removed, there was a liberation of iodine which could only be removed by continued boiling to expel the iodine before the colour could be developed. If much iron were present, phosphoric acid must be substituted for part of the sulphuric acid used, to prevent the development of any yellow colour due to iron.

Preparation of Solution

In preparing the solution for the final manganese determination, wet-oxidation of the tissues was preferred. The possibility of loss due to fusion of particles, as may occur in the ashing process, is minimized. In addition, there is the additional advantage that the final solution can be used for the determination of other metals. For example, in the present work, the solutions used were those from which an aliquot had already been taken to determine the arsenic present in the tissues.

The tissues were minced, when possible, or cut into fine pieces when mincing was not practicable. The required weight of tissue, usually 50 gm., was treated in a Kjeldahl flask with about 100 ml. of 10% nitric acid. The contents of the flask were heated, gently at first and later to boiling, until the mass had dissolved, leaving only a layer of unoxidized fat (if any) on the surface of the liquid. The solution was then cooled and filtered into a second Kjeldahl flask, and the residue of fat boiled with a further small amount of dilute nitric acid. This second solution was then filtered and added to the first solution; 10–15 ml. of concentrated sulphuric acid were now added to the solution and the whole boiled down until the mixture began to darken. Sufficient concentrated nitric acid was added to the *hot* solution to clear the mixture and the boiling continued. The addition of nitric acid was repeated whenever necessary until finally the solution remained clear and white fumes began to appear. When manganese was to be determined, it was not essential to remove the last traces of nitric acid. The solution was therefore cooled and made up to a final volume, usually 50 ml.

In the case of human tissues 10–20 gm. are sufficient for the actual determination. If this quantity of tissue be used the amount of concentrated acid used could be reduced correspondingly. The use of the larger weight of tissue, however, has the advantage of obtaining a more representative sample, especially when the tissue is not uniform in structure.

Method of Determination. For the determination 20 ml. of the above prepared solution were transferred to a silica basin and evaporated on a sand bath until no further white fumes were produced. The residue was then heated over a flame until the last traces of sulphuric acid were expelled and all remaining organic matter was destroyed, leaving a clear residue. After cooling, the residue was treated with 10 ml. of water containing 1 ml. of a mixture of approximately equal parts of concentrated sulphuric acid and 80% phosphoric acid. The solution was then transferred to a 6″ x 1¼″ Pyrex test-tube and the silica basin washed with two further 5 ml. lots of water, containing ½ ml. of the mixed acids per 10 ml. If necessary, a further few ml. of water were used to complete the transfer of the solution. If the solution were unduly cloudy it was filtered to remove the suspended materials; if it were only slightly cloudy, no further action was taken since; during the subsequent heating with periodate, the suspended matter settled to the bottom of the tube, and the clear solution poured off.

About 0.2–0.3 gm. of potassium periodate were now added to the solution and the mixture brought to the boil over a flame. The tube was then placed in a boiling water-bath and allowed to remain until the full colour had developed. During the heating the tube was not covered and the solution was allowed to concentrate so that the acidity reached the most suitable concentration for colour development.

After the maximum colour had developed, the solution was transferred to one of a pair of matching tubes and a slightly less volume of water was placed in the second tube. N/1000 potassium permanganate was added to the second tube from a burette until a colour match was obtained, care being taken that the final volumes of the solutions were as nearby as possible the same in both tubes. 1 ml. of N/1000

potassium permanganate is equivalent to 0.011 mgm. of manganese. If necessary, the unknown solution could be re-heated in the water-bath to ensure that the colour had fully developed.

It was found that the ordinary clear glass flat-bottomed specimen tubes formed very suitable matching tubes. They were readily standardized by adding known volumes of water from a burette and marking them at suitable intervals by means of a file.

For purposes of checking the accuracy of the method, a solution of manganese sulphate was prepared, containing 0.01 mg. of manganese per ml. Known amounts of this solution were oxidized by the above method and the amounts of manganese present determined. In other cases, known amounts were added to solutions prepared from various tissues after the manganese content of the tissues had been determined. Table I gives the results obtained:

Table I

(Amounts of Manganese expressed in mg.)

Solution	*Amount Added*	*Amount Found*	*Amount Recovered*
Water	0.0250	0.0242	0.0242
„	0.0250	0.0253	0.0253
„	0.0250	0.0242	0.0242
Liver	—	0.0319	—
„	0.0100	0.0429	0.0110
Kidney	—	0.0132	—
„	0.0275	0.0402	0.0270
„	—	0.0121	—
„	0.0100	0.0237	0.0116
Stomach	—	0.0055	—
„	0.0150	0.0210	0.0155
Intestines	—	0.0077	—
„	0.0200	0.0275	0.0198
Potatoes	—	0.0462	—
„	0.0300	0.0759	0.0297
	Total 0.1875 mgm.		0.1883 mgm.

It will be noted that the recovery is excellent and that the method is entirely satisfactory. It has been used to determine the normal manganese content of tissues such as hair and nails and of blood, faeces and urine.

References

1. Monier-Williams, G. W. (1949): *Trace Elements in Food.* London: Chapman and Hall, Ltd.
2. Richards, M. B. (1930): *The Colorimetric Determination of Manganese in Biological Material,* Anal., **55**, 554.
3. Snell, F. D. and Snell, C. T. 1937): *Colorimetric Methods of Analysis.* London: Chapman and Hall, Ltd.
4. Willard, H. H. and Greathouse, L. H. (1917): *The Colorimetric Determination of Manganese,* J. Amer. Chem. Soc., **39**, 2366.

REVIEWS OF BOOKS

Medico-Legal Pathology and Toxicology

Legal Medicine, Pathology and Toxicology. By Thomas Z. Gonzales, M.D., Morgan Vance, M.D., Milton Helpern, M.D., and Charles J. Umberger, Ph.D. (Pp. 1349 + xii, with numerous illustrations. 2nd. ed. $22.00.) New York: Appleton-Century-Crofts, Inc. 1954.

Contents: 1. The Medical Examiner and the Coroner 2. Investigation at the Scene of Death. 3. Identification. 4. The Signs of Death. 5. The Medicolegal Autopsy; Determination of the Cause of Death. 6. Unexpected and Sudden Natural Death. 7. Natural Death and Trauma. 8. Types and Complications of Trauma. 9. Blunt Force Injuries of the Skin and Subcutaneous Tissues. 10. Blunt Force Injuries of the Extremities. 11. Blunt Force Injuries of the Chest and Abdomen. 12. Blunt Force Injuries of the Head. 13. Blunt Force Injuries of the Neck and Spine. 14. Correlation of the Type of Blunt Force with the Injuries. 15 Stab Wounds: Incised Wounds: Chop Wounds: Wounds Produced by Foreign Bodies: Biting, Scratching and Gouging. 16. Bullet and Other Missile Wounds. 17. Traumatic Asphyxia: Strangulation. 18. Traumatic Asphyxia: Suffocation; External Pressure on Chest; Drowning. 19. Asphyxiating Gases. 20. Thermic Trauma: Electricity: Roentgen Rays: Starvation: Atmospheric Pressure. 21. Pregnancy: Puerperium: Illegitimacy. 22. Abortion. 23. Infanticide. 24. Verginity: Rape and Other Sexual Assaults: Examination of Semen. 25. Impotence: Sterility: Hermaphrodism: Monstrosities. 26. Examination of Blood. 27. The Human Blood Groups. 28. Microscopy of Hair and Other Material. 29. Toxicology: General Considerations. 30. Inorganic Poisons: Corrosives. 31. Inorganic Poisons: Metals and Nonmetals. 32. Organic Poisons: Volatile. 33. Organic Poisons: Nonvolatile, Nonalkaloidal. 34. Organic Poisons: Alkaloids. 35. Miscellaneous Poisons. 36. Occupational Diseases and Toxicology. 37. The Government and the Physician. 38. The Rights and Obligations of Physicians: Malpractice. 39. Insanity. 40. Insurance and Survivorship: Malingering. 41. Analytic Toxicology: Introduction. 42. Analytic Toxicology: Gaseous Poisons. 43. Analytic Toxicology: Inorganic Metallic Poisons. 44. Analytic Toxicology: Inorganic Nonmetallic Poisons. 45. Analytic Toxicology: Inorganic and Organic Poisons Volatile with Steam. 46. Analytic Toxicology: Ethyl Alcohol. 47. Analytic Toxicology: The Nonvolatile Organic Poisons. Isolation and Extraction. Preliminary Examination, Purification Method, Sedative Identification. Identification Process.
Appendix. Index.

This volume is a distinguished contribution to the literature of forensic medicine. It derives an unusual authority from the vastness of the personal experience of each of the contributors. This makes the book one of the most practical available, providing an encyclopaedic reference source for the whole field of traumatic pathology. Some 80-90% of the book is thus devoted to problems that are the daily concern of the medico-legal pathologist, who will find the material presented in a detail not readily equalled. Especially valuable is the profusion of pertinent photographs.

The soundness with which fundamental observations about the dead are presented should be an object lesson to those concerned with this field of practice. There is a remarkable lack of appreciation, amongst many practitioners, of the diversity of the consequences that follow from the effects of gravity on the dead body. Those wedded to the asphyxial significance attributed to small haemorrhages in the skin of the face, neck and shoulders would do well to ponder the statement that 'the pressure of gravity may cause postmortem rupture of some of the smaller blood vessels, producing a minute circular haemorrhage known as a Tardieu spot. These lesions are found most frequently in the loose connective tissues of the neck and shoulders.

If the body is lying in the prone position, face down . . . Tardieu spots are common. Sometimes the congestion is so great that minute blood vessels are ruptured in the nose, and an appreciable flow of blood will occur from the nasal orifice' (p. 52). While these postmortem haemorrhages are usually discrete, they may also become confluent (Fig. 4-2, p. 52).

This alert series of fundamental observations confirms the experience gained by others and invites a considerable caution in the postmortem diagnosis of asphyxial death from cadaveric signs alone.

It is, however, surprising that the authors repeat the unwarranted assertion that the blood in the heart in a typical asphyxial death is dark and fluid. It is widely recognized to-day that the liquidity of the blood after death conveys no clue about the mode of death, asphyxial or otherwise, since unoxygenated blood does not influence the fluidity of the blood.

Although the authors do not accept wholly the hypothesis that status lymphaticus is an obvious and ready

explanation for obscure and sudden deaths, they extend the concept considerably in being prepared to attribute sudden death from subarachnoid or cerebral haemorrhage to rupture of a hypoplastic cerebral artery. (They follow the orthodox convention of regarding lack of development of the heart and circulatory system as something associated with underdeveloped adrenal glands and generalized enlargement of lymphatic tissues). On p. 148 a case of sudden death from subarachnoid haemorrhage during a boxing match is thus explained on a basis of status lymphaticus. This opinion is likely to be controverted very widely.

The acceptability of status lymphaticus as an entity has recently again been challenged by Geoffrey Keynes (Brit. Med. J., 1954, **2**, 659) who emphasized that the thymus does not disappear with age, that its size is inconstant and that it is always there. Yet the problem may have been oversimplified, as Keynes himself admits that he has abolished myasthenia gravis in patients from whom he has removed an apparently normal thymus gland (not persistent or enlarged by his own criteria); and Selye's work on the adrenals and the thymus strongly supports the view that sudden and unexpected death from trivial causes may be related to some glandular mechanism connected with the thymus.

Although justifiable criticisms may be offered here and there, these must be regarded as minor when one considers the massive accomplishment the authors have achieved in this magnificent volume. The inclusion of an adequate section on toxicology enhances the utility of a work which has acquired a pre-eminent place in the forensic field.

Forensic Psychiatry

Forensic Psychiatry. By Henry A. Davidson, M.D. (Pp. 387. $8.00.) The Ronald Press Company, New York.

Contents: Part I: The Content of Forensic Psychiatry: 1. Criminal Responsibility. 2. Examination of the Defendant. 3. Reporting the Criminal Case. 4. Personal Injury Evaluation. 5. Marriage, Divorce and Annulment. 6. Placement and Custody of Children. 7. Last Will and Testament. 8. Appraisal of the Sex Offender. 9. Medico-Legal Aspects of Alcoholism. 10. The Psychiatrist and the Juvenile Court. 11. Malingering. 12. Commitment Procedures. 13. Competency, Contracts and Civil Rights of the Mentally Ill. 14. Malpractice and the Psychiatrist.

Part II: The Tactic of Testimony: 15. The Nature of Medical Testimony. 16. Qualifying the Psychiatric Expert. 17. Preparation for Court Appearance. 18. Office and Hospital Records. 19. Direct Examination of the Medical Witness. 20. That Hypothetical Question. 21. The Translation of Technical Terms. 22. Cross-Examination. 23. Courtroom Etiquette.

Appendix: A. Legal Lexicon for Doctors. B. Psychiatric Glossary for Lawyers. C. Examination Guides. D. Model Act for the Certification of the Mentally Ill.

Psychiatric texts intended for medico-legal reference are generally a simplified presentation, along orthodox lines, of the material usually covered in standard works. They do not provide for the pitfalls and the difficulties the conscientious medical witness is likely to encounter once he enters the witness box. Indeed, there is a serious hiatus in forensic writings from this point of view, because the few articles which might be helpful in this regard are scattered widely throughout the periodical literature.

Dr. Davidson has made a radical departure from the custom of most authors in the field of forensic psychiatry. He has written what is probably a unique book which deals specifically with those aspects of psychiatry which bring doctor and lawyer together in the courtroom. It must be one of the few books, if not the only one, of its kind.

Clearly based on a wealth of direct personal experience from which the author has gained many practical lessons, the volume will be a welcome guide to the practitioners of both professions involved. Particularly useful is the extremely logical way in which problems in forensic work are presented, as well as the comprehensive classifications suggested for their solution. This is, e.g. very satisfactorily brought out in the particular case of testamentary capacity, where the threefold requirements fundamental for the validity of a will are stated in such a sound way as to become an excellent guide for all stages of dealing with this problem, both before and after the death of the testator.

The discussion of criminal responsibility which opens the book (together with the presentation of the concept of the irresistible impulse) is one which will be intelligible to lawyers as well as doctors. The balanced appraisal of the M'Naghten Rules makes it clear that the Rules *are* adequate for the contingencies with which they were meant to deal, and that they cannot be stretched in a Procrustean fashion to cover eventualities with which they were never intended to cope.

The vexed problem of the psychopath and his responsibility is reviewed in all its varied aspects. The concept of diminished responsibility (which has in recent decades been finding favour in Scottish courts) is apparently likely to receive scant sympathy in the U.S.A. It is true that the psychopath cannot be excused merely because he suffers from an illness. As Dr. Davidson points out, this is tantamount to saving that a blind man should not be denied a driver's

licence, because his blindness is not his fault. Yet there seems room to judge each psychopathic case on its merits, since not all psychopaths are of the same grave degree. Moreover, although the technicality of the guilt might well not be disputed in a particular trial, the question of sentence could reasonably come under review, particularly in capital cases. An enlightened penal system would eschew a primitive optico-dental approach to punishment, and truly make the punishment fit the crime under the conditions in which it was committed.

The difficult problem of malingering is generally disposed of in a few paragraphs. It is characteristic of the thoroughness with which this volume has been prepared that this subject rates a complete chapter on its own.

Most important, perhaps, is the care taken in presenting for the practitioner the *Tactics of Testimony*. No potential medical witness can afford to neglect a close study of this section of the book. The treatment of the technique of cross-examination and the intricacies of the hypothetical question makes for a prepared witness with an intelligent appreciation of what will be required of him.

This helpful contribution to medical practice in our courts is assured of an enthusiastic welcome.

Disputed Paternity

Disputed Paternity Proceedings. By Sidney B. Schatkin, Assistant Corporation Counsel of the City of New York. (Pp. 823, 3rd ed.) New York: Matthew Bender and Company Inc., 1953.

Contents: Part I. *The Law and Practice in Affiliation. Rules of Evidence, Including the Decisive Evidence of Non-Paternity Furnished by Blood Tests.* 1. The Natural Child. 2. Nature of the Affiliation Proceeding. 3. Rules of Evidence Identified with Affiliation Proceedings. 4. The Scientific Aspect of Blood Tests. 5. The Rh-Hr Blood Types. Part I. 6. The Rh-Hr Blood Types. Part 2. 7. The Legal Aspect of Blood Tests. 8. Blood Tests—An Effective Rebuttal of the Presumption of Legitimacy. 9. The Unerring Accuracy of Blood Tests. 10. Compromise and Settlement. 11. Paternity Proceeding on Behalf of Foreign-Born Child against American Citizen Residing in the United States. 12. Order of Filiation. 13. The Illegitimate Child's Birth Certificate. Part II. *Defending the Proceeding.* 14. Points on the Conduct of an Affiliation Trial. 15. The Duration of Pregnancy: Its Bearing on the Ellusive Question, "Is the Defendant the Father of the Child?" 16. Cross-Examination of the Mother.

Appendices. 1. New York City Criminal Courts Act, Article V. 2. Domestic Relations Law, Article VIII. 3. Uniform Illegitimacy Act. 4. Summary of Affiliation Statutes Throughout the United States. 5. Forms.

As Assistant Corporation Counsel of the City of New York Mr. Schatkin has had a unique experience of disputed paternity proceedings. His carefully documented review of this problem supersedes the necessarily limited accounts to be found in medico-legal journals or textbooks. His definitive survey gives guidance to the lawyer on procedural as well as all the other legal aspects involved in these suits.

Blood tests for the exclusion of paternity have had (and are still having) difficulty in achieving adequate recognition in all the American courts. The farcical situation that can arise (with great injustice to the defendant) is well exemplified in the Charles Chaplin case, which is described fairly fully.

Mr. Chaplin was group O (type MN); the mother was group A (type N); and the child was group B (type N). Although Mr. Chaplin could not be excluded on the MN system, he was wholly and unequivocally excluded on the ABO system. Nevertheless the California court (bound by the 'bitterly criticized case of Arais *v.* Kalensnikoff (1937), 74P (2d) 1043,' found Mr. Chaplin to be the father on the non-medical and non-scientific evidence. This included an allegation by the mother that she confronted Mr. Chaplin in his home on one of the material nights with a loaded gun. This she retained in her hand throughout the interview, 'except for an interlude when they indulged in sexual intercourse, on the completion of which act, she again took up the gun' (p. 258).

Mr. Schatkin rightly points out that the 'Chaplin verdict is contrary to science, nature and truth. The verdict bears no relation to fact. The position of the California Supreme Court that blood tests are the subject of experts' conflicting opinions, is incorrect . . . Inasmuch as the Chaplin verdict received wide publicity, it is apt to create a doubt in the public mind, as to the correctness of blood test exclusions' (p. 256).

The complainant's counsel of course had no difficulty, on cross-examination, in obtaining from the serologist the admission that, on the MN system, Chaplin could have been the father. This illogic is tantamount, suggests the author, to arguing for the discharge and acquittal of an accused person because the prosecutor has only one witness who saw him commit the crime and the accused 'can produce one hundred witnesses who did not see him do the act' (p. 262).

It is significant that this highly misleading type of cross-examination is not permitted in Denmark, where inconclusive data are omitted from the report, if paternity is conclusively incompatible with any of the blood group systems employed.

The volume has much of interest on the vexed problem of artificial insemination which can undoubtedly be held to constitute adultery and rape.

There is also a useful discussion of the duration of pregnancy, which American courts have accepted to last as long as 355 days (p. 522). English courts have accepted 346 days (Wood *v.* Wood, 1947, P. 103; 1947, 2 All E.R. 95) and 349 days (Hadlum *v.* Hadlum, 1949, P. 197; 1948, 2 All E.R. 412), but they have boggled at adding on another 11 days. The House of Lords, e.g. rejected the alleged figure of 360 days (Preston-Jones *v.* Preston-Jones, 1951, A.C. 391; 1951, All E.R. 124).

Judicial gullibility has undoubtedly been aided by somewhat credulous medical reports published from time to time, some of them virtually apocryphal.

Stewart (J. Amer. Med. Assoc., 1952, 148, 1079) has recently published a salutary review of this problem. He studied 135 women for 5 years, using their oral basal temperature to establish the date of ovulation. The pregnancy duration is not therefore calculated from the last menstrual period. He concludes that there is no evidence to support the prolongation of human pregnancy to 10, 11 or 12 months. In his series, pregnancies lasted on the average 266-270 days, the maximum period being 285 days. The date of the last menstruation is quite misleading, as ovulation may be suppressed or delayed.

Mr. Schatkin's contribution should be in the library of every forensic practitioner, legal or medical. His expert marshalling of the persuasive and compelling evidence will do much to establish the acceptability of blood group tests in courts which look askance and with suspicion on this unbiased, objective and disinterested testimony. Indeed, he may be regarded as having already succeeded, in part, in this objective in the United States.

An Anthology of Libel

Hatred, Ridicule or Contempt. By Joseph Dean. (Pp. 271, 15s.). London: Constable and Company Limited, 1954.

Contents: 1. Salome and the Black Book. 2. Winston Churchill and the Battle of Jutland. 3. The Ordainments of the Theatre. 4. Sermons in Trances. 5. Vocal Sins. 6. The Reputation of the Dead. 7. Libel or no Libel. 8. Dead or Alive. 9. Arthemus Jones and his Consequences. 10. Two Stockbrokers. 11. Visual Images. 12. A Rose by any Other Name. 13. Rasputin. 14. A Policeman's Feet. 15. A Dramatic Dance. 16. Bottomley's Last Case. 17. Blackmail. 18. Black Magic. 19. The Westminster Libel Shop. 20. Surgery in 1828. 21. Revolution by Violence. 22. The Last Trial in Westminster Hall.

It is not surprising that this unusual anthology had such success on publication as to require a reprint very soon afterwards.

It is a collection of memorable libel actions, and because Mr. Dean has excluded the more hackneyed trials, the cases reported impress the reader with a certain novelty and freshness.

Mr. Dean's technique of editing relies on the extensive verbatim quotation of the verbal duels between witness and counsel. The volume therefore provides a record of outstanding examples of cross-examination, whether surgeons and their surgery are being dealt with in 1828 or Mr. Harold Laski is being discomfited by Sir Patrick Hastings in 1946.

We have greater freedom with our tongues than our typewriters, and the actions reported illustrate the way in which libel laws protect the individual and not the group. Indeed, this provides the basis whereby much of our freedom in public discussion is preserved. 'Libel holds the scales of justice between the protection of private reputation on the one hand and public freedom of speech on the other. Scales so weighted are not easily balanced.'

In the course of ensuring this precarious balance the notable forensic skill of the protagonists has provided a series of occasions which will delight the lay as well as the professional reader.

CORRESPONDENCE

Breath Tests for Alcohol

To the Editor: I am wondering if your editorial, *Breath Tests for Alcohol*, which appeared in the July-September, 1954 issue of the *Journal*, was perhaps purposely biased in order to stimulate discussion from north of the equator. If so, I will try to oblige you, because this editorial abounds in misinformation.

You refer, in a somewhat disparaging tone, to the recent report on breath alcohol tests[1a] by the Committee on Tests for Intoxication of the National Safety Council (U.S.A.), although you have apparently read only an abstract of this report. Enclosed you will find a copy of the complete report. I hope you will study it carefully and then give your readers the salient points in it. As you will see, this study of breath: blood results was made by an impartial, qualified sub-committee, the members of which had no personal interest in any of the three breath methods under investigation. *The report gives individual results, not statistical averages, and the conclusions are based on the former**.

As regards specificity for alcohol, the breath methods are actually superior to most blood methods, because distillation at elevated temperatures could conceivably bring over oxidizable substances which are not volatile at body temperature. With the drunkometer acetone causes no interference whatever as is also true for many organic solvents used in industry. As a matter of fact, with both breath and blood methods, the likelihood of a false positive result is extremely remote.

You quote from Dr. Rabinowitch. He has repeatedly condemned breath methods, without even bothering to try them out personally. Blood methods find little favour with him, either. To show the fallacy of some of his arguments, I suggest that you read a 1948 article by him[1] and also replies by Dr. Muehlberger[2] and by myself[3].

In spite of objections from a few armchair critics, these breath methods do produce results which are amply accurate for practical purposes. In addition to the National Safety Council report, recent studies by Mr. James Osterburg of the New York City police laboratory[4] and by Dr. H. Ward Smith of the University of Toronto[5] confirm the reliability of these breath methods.

I will now comment on your eight items from which you allege breath tests are 'severely vitiated'.

1. *Alcohol Residues in Mouth.* This is a threadbare alibi of some defence attorneys. In our 1938 paper[6] we stated:

'As pointed out by Bogen, nonalcoholic subjects who simply rinse out the mouth with a strong alcoholic beverage such as whiskey will exhibit alcohol in the breath for a short time afterward. However, as shown by Bogen and confirmed by ourselves, this effect is almost gone in five minutes and entirely disappears after about ten minutes. For this

* The Tables published in this Report disclose a range of deviation which is specifically stated on pp. 7, 8 and 9 together with the average deviation for each of the three methods used.

The range of deviation is as follows:
For the Intoximeter: −0.016% to +0.020%.
For the Alcometer: −0.020% to +0.021%.
For the Drunkometer. −0.020% to +0.028%.
These are salient features (somewhat contaminated, it is true, by statistical treatment) and glossed over by our correspondent.—*Editor.*

reason a period of at least ten minutes should elapse between the taking of alcoholic fluids into the mouth and the collection of the breath. In the practical testing of automobile drivers this source of error seems unimportant, but it should certainly be recognized by persons using the method'.

Alcohol applied locally in the mouth disappears quickly because it is highly diffusible and is absorbed by all mucous surfaces. You might test this out for your own satisfaction.

2. *Sucking Menthol-Containing Pastilles.* Over the years 'debunkers' of the drunkometer have suggested numerous substances which they claimed would give false results with this test. As a result of these claims we have tested onions, garlic, odoriferous cheese, sen-sen, citrus fruit rinds, etc. The results were uniformly negative[7]. So, with some weariness, we purchased two popular brands of menthol lozenges and also two types of menthol inhaler. In testing the lozenges a subject held two in his mouth for 15 minutes, and with the inhalers he inspired deeply through them 10 times. Immediately after each type of menthol administration we ran the subject's breath through the purple drunkometer fluid. The colour remained unchanged even after the passage of six liters of this breath. For a person with a blood alcohol concentration of 0.15% the permanganate would have been entirely decolorized by about 360 c.c. of his breath. We next tested air forced directly through the menthol inhalers, with the same negative result. You did not identify the author of this menthol rumor, but I assume that it was not the respected editor of this *Journal.*

3. *Belching During Collection of Breath Samples.* I suggest that you try to belch while inflating a rubber balloon. The air pressure required, about 20 inches of water, will insure closure of the esophagus. I have run thousands of breath tests and have yet to observe the first belch during the collection of the sample. Moreover, the alcohol level of the stomach rapidly falls and approaches that of the blood within an hour after drinking, at which time stomach gas and breath will have about the same concentration of alcohol.

4. *Report of Deviation from the Accepted Blood : Breath Ratio Shortly after Drinking.* Dr. Goldberg's personal communication on this matter probably referred to a 1930 paper by his mentor, Dr. Liljestrand[8], who conducted blood : breath studies *using peripheral venous blood.* He reported that, for about an hour after drinking, the breath levels were somewhat higher than the uniform blood : breath ratio of about 2000 : 1 observed later. Subsequently, both Haggard and Greenberg of Yale[9], and ourselves[10], have shown that, during the period of active absorption, peripheral venous blood lags behind arterial blood as regards alcohol level. We have shown[11] that the alcohol level of the brain always closely parallels that of heart blood, even as early as 10 minutes after drinking. It is also heart blood which controls the level of alcohol in the breath. Therefore, during the absorption period, breath is actually superior to peripheral venous blood for predicting the alcohol level of the brain. We stated this in a 1950 paper[12]. It is generally accepted that capillary blood has practically the same alcohol level as arterial blood and that rebreathed air is essentially alveolar air as regards alcohol content. In a study completed during the past summer we compared the alcohol level of rebreathed air with the levels in capillary blood and in peripheral venous blood.

The time intervals between the drinking and the analyses ranged from 10 minutes to 3 hours. At all times the capillary blood : rebreathed air ratio was close to 2100 : 1, but during the first hour after drinking, the peripheral venous blood frequently lagged behind the capillary blood by as much as 15 to 20%. This means that, shortly after drinking, peripheral venous blood, and not breath, is of questionable reliability.

5. *Lack of Training.* Prof. Erik Wolff deplores the operation of the drunkometer by 'untrained persons'. So do we[13]. However, the correct operation of the drunkometer is a very simple matter and an intelligent police officer can be taught in a few days to operate the device in a reliable manner. We will probably soon change to rebreathed air, which is even simpler as it eliminates the use of an analytical balance. Our recent studies showed that securing cooperation by the subject in rebreathing is not as difficult as one might

imagine. Some of our subjects were too drunk to even drive a car, but did the rebreathing well.

6. *Effect of Depth of Breathing.* Depth of breathing does affect the concentration of alcohol in the breath. It also affects the concentration of carbon dioxide in the breath to the same extent. That is the reason for making simultaneous analyses for alcohol and carbon dioxide, when using the drunkometer or intoximeter. This procedure enables one to use ordinary expired air rather than having to obtain alveolar air. The National Safety Council Committee found that the use of this breath alcohol : carbon dioxide ratio will reliably predict the alcohol level of the blood.

7. *Recent Report by British Medical Association.* I have a copy of this report and have read it carefully. Although the Scandinavian countries and some other European nations have been using blood analyses as evidence of intoxication for over 20 years, England has consistently held aloof from these tests. This latest report does seem to sanction the use of blood, at least in a measure. Perhaps in another ten years England may see the light as regards breath tests.

8. *Rabinowitch again, on 'Statistical' Evidence.* You have quoted from both Rabinowitch and Goldberg. Which do you consider an authority? Rabinowitch apparently claims that *no* level of blood alcohol is high enough to convict a given individual beyond a reasonable doubt. Goldberg asserts that *all* persons with a blood alcohol above 0.05% show definite impairment of driving ability, with all seriously impaired above 0.10%, and his careful studies[14] confirm his views. Most of us here agree with Goldberg.

References

1. Rabinowitch, I. M. (1948): '*Medicolegal Aspects of Chemical Tests for Intoxication*', J. Crim. Law Criminol., **39,** 225.

1*a*. Committee on Tests for Intoxication, National Safety Council (1953): '*Evaluating Chemical Tests for Intoxication*', National Safety Council, 425 North Michigan Ave., Chicago.

2. Muehlberger, C. W. (1948): *Comments on Dr. Rabinowitch's Paper*, J. Crim. Law Criminol., **39,** 411.

3. Harger, R. N. (1948): *Comments on Dr. Rabinowitch's Paper*, J. Crim. Law Criminol., **39,** 402.

4. Osterburg, J. W. (1954): Personal Communication.

5. Smith, H. Ward (1954): Personal Communication.

6. Harger, R. N., Lamb, E. B. and Hulpieu, H. R. (1938): *A Rapid Chemical Test for Intoxication Employing Breath*, J. Amer. Med. Assoc., **110,** 779.

7. Harger, R. N. (1949): *Debunking the Drunkometer*, J. Crim. Law Criminol., **40,** 497.

8. Liljestrand, G. and Linde, P. (1930): *Uber die Ausscheidung des Alkohols mit der Expirationsluft*, Skandinav. Arch. fur Physiol., **60,** 273.

9. Haggard, H. and Greenberg, L. A. (1934): *The Concentration of Alcohol in Arterial Blood and in Venous Blood from Different Parts of the Body*, J. Pharmacol. Exptl. Therap., **52,** 157.

10. Harger, R. N., Hulpieu, H. R. and Cole, V. V. (1945): *The Ratio of the Concentration of Alcohol in Heart Blood and in Blood from Peripheral Veins*, Federation Proceedings, **4,** 123.

11. Harger, R. N. (1951): *The Correlation Between the Concentration of Alcohol in the Brain and that in Arterial Blood and Venous Blood at Various Time Intervals Following the Administration of Alcohol*, Proc. Amer. Acad. Forensic Sciences, **1,** 164.

12. Harger, R. N., Forney, R. B. and Barnes, H. H. (1950): *Estimation of the Level of Blood Alcohol from Analysis of Breath*, J. Lab. Clin. Med., **36,** 306.

13. Harger, R. N. (1944): *Some Practical Aspects of Chemical Tests for Intoxication*, J. Crim. Law Criminol., **35,** 202.

14. Bjerver, K. and Goldberg, L. (1950): *Effect of Alcohol Ingestion on Driving Ability*, Quart. J. Stud. Alcohol, **11,** 1-30.

R. N. Harger,
Professor of Biochemistry and Toxicology

Indiana University School of Medicine,
Indianapolis,
Indiana, U.S.A.

21 September, 1954.